Environment Canada Environnement Canada

Canadian Water Quality Guidelines for Carbofuran

D.M. Trotter,* R.A. Kent, and M.P. Wong

*Monenco Consulting Ltd.
Calgary, Alberta

SCIENTIFIC SERIES NO. 169

INLAND WATERS DIRECTORATE
WATER QUALITY BRANCH
OTTAWA, ONTARIO, 1989

(Disponible en français sur demande)

90-03121/

Published by authority of
the Minister of the Environment

©Minister of Supply and Services Canada 1989

Cat. No. En 36-502/169E

ISBN 0-662-17142-X

Contents

	Page
ABSTRACT	v
RÉSUMÉ	v
SOURCES, OCCURRENCE, AND CHARACTERISTICS	1
Uses and production	1
Chemical characteristics	2
Carbofuran in the environment	2
Persistence and degradation	3
RATIONALE	6
Raw water for drinking water supply	6
Guideline	6
Freshwater aquatic life	6
Levels and fate in the aquatic environment	6
Accumulation and elimination of carbofuran in aquatic biota	9
Acute toxicity to aquatic organisms	9
Fish	9
Invertebrates	10
Plants	10
Sublethal reactions and chronic toxicity in aquatic organisms	10
Fish	10
Invertebrates	13
Plants	13
Guideline	14
Agricultural uses	14
Livestock watering	14
Acute toxicity to livestock	14
Subacute and chronic toxicity to livestock	17
Uptake, metabolism, and depuration	18
Mutagenicity, teratogenicity, and carcinogenicity	19
Guideline	20
Irrigation	20
Guideline	20
Recreational water quality and aesthetics	20
Guideline	20
Industrial water supplies	20
Guideline	20
SUMMARY	20
ACKNOWLEDGMENTS	20
REFERENCES	20
APPENDIX A. Summary of carbofuran residues found in aquatic environments	25
APPENDIX B. Carbofuran acute toxicity values for aquatic organisms	29

Tables

		Page
1.	Carbofuran sales in Canada	1
2.	Physical and chemical properties of carbofuran	2
3.	Loss of carbofuran from paddy and pond water in the field	4
4.	Acute oral toxicities of carbofuran to birds and mammals	15
5.	Acute toxicity of dietary carbofuran to birds and mammals	16
6.	Recommended water quality guidelines for carbofuran	20

Illustrations

Figure 1. Structural formula for carbofuran 1

Abstract

A literature review was conducted on the uses, fate, and effects of carbofuran on raw water for drinking water supply, freshwater aquatic life, agricultural uses, recreational water quality and aesthetics, and industrial water supplies. The information is summarized in this publication. From it, water quality guidelines for the protection of specific water uses are recommended.

Résumé

On a examiné la documentation relative aux utilisations, au devenir et aux effets du carbofuran sur l'eau naturelle utilisée comme eau potable non traitée, sur la vie aquatique en eau douce, sur l'utilisation de l'eau pour l'agriculture, sur la qualité de l'eau pour les loisirs et l'esthétique, ainsi que sur les approvisionnements en eau pour l'industrie. Ces renseignements sont résumés dans cette publication. À partir de cette étude, des lignes directrices sur la qualité de l'eau sont recommandées pour la protection d'utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Carbofuran

D.M. Trotter, R.A. Kent and M.P. Wong

SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Carbofuran is the common name for the chemical 2,3-dihydro-2,2-dimethylbenzofuran-7-yl methylcarbamate (IUPAC) or 2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate (C.A.). The structural formula of carbofuran is shown in Figure 1. It was originally produced in 1969 by the Agricultural Chemical Division of FMC Corporation under U.S. Patents 3474170 and 3474171. Its Chemical Abstracts Service Registry Number is 1563-66-2. Other names and registered trademarks include Furadan^{R}, Curaterr^{R}, Yaltox^{R}, Bay 70143, NIA 10242, and ENT 27164 (Thomson, 1979; Worthing and Walker, 1983).

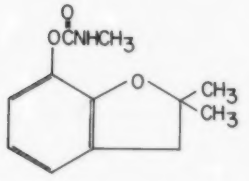

Figure 1. Structural formula for carbofuran.

Carbofuran is a major use systemic pesticide in Canada. Sales data from a 1982 retailer survey conducted by Monenco Ltd. (1984) for the Canadian Wildlife Service and additional data collected in 1986 are presented in Table 1. A survey of the various provincial agricultural and environmental ministries in 1986 indicated that British Columbia, Saskatchewan, and Manitoba did not keep records concerning carbofuran use within their respective jurisdictions. During the summer of 1986, the government of Nova Scotia approved legislation requiring the licensing of pesticide vendors and the annual reporting of pesticide quantities sold. Prior to this legislation, no records were kept by the government of Nova Scotia concerning pesticide sales or use. The quantities of pesticides sold in Quebec are reported to the Ministry of Environment and Environment Canada on a voluntary basis only and no figures are available for carbofuran alone. The only reporting of carbofuran use in Alberta is through the Alberta Farm Grasshopper Control Assistance Program. This program reimburses farmers for the purchase of insecticides. Records are kept regarding the litres of product sold by district, the cost per district, and the average cost per farm.

Table 1. Carbofuran Sales in Canada

Province	Year	Quantity*
New Brunswick	1982	2943 kg active ingredient
	1985	2278 kg + 2122 L of product
	1986	1140 kg + 1322 L of product
Prince Edward Island	1982	1100 kg + 2120 L of product
Nova Scotia	1982	7065 kg + 527 L of product
Newfoundland	1982	5960 kg + 792 L of product
Ontario	1983	43 890 kg active ingredient
Alberta	1985	123 756 L of product
	1986	130 361 L of product

Source: Monenco Ltd., 1984; McGee, 1984; Brown, 1987, New Brunswick Ministry of Agriculture, pers. com.

*Information on specific product formulation sold was not available. Provinces vary in reporting requirements (i.e., quality of active ingredient versus quantity of commercial product).

Carbofuran is a systemic acaricide, insecticide, and nematicide, which may be applied to foliage at 0.25–1.0 kg-ai-ha^{-1} (ai = active ingredient). It may also be applied to seed furrows at 0.5–4.0 kg-ai-ha^{-1} or broadcast at 6–10 kg-ai-ha^{-1} (Worthing

and Walker, 1983). Carbofuran has been used on the following crops (Worthing and Walker, 1983):

corn	sugar beets
alfalfa	peanuts
mixed vegetables	peppers
canola	strawberries
onions	rutabagas
tobacco	turnips
mustard	sorghum
carrots	potatoes
sunflowers	raspberries

The following pests are controlled by carbofuran (Worthing and Walker, 1983):

corn rootworms	wireworms
alfalfa weevils	boll weevils
aphids	scale
thrips	European corn borers
mosquitoes	army worms
nematodes	grasshoppers

Carbofuran formulations include 2%, 3%, 5%, and 10% ai in granules, 480 g-ai·L^{-1} as a flowable paste, and 750 g-ai·kg^{-1} as a wettable powder.

In the Atlantic provinces of New Brunswick, Newfoundland, and Prince Edward Island, carbofuran is used primarily on potatoes. A user survey in Nova Scotia indicated carbofuran was used on mixed vegetables. The granular formulations are banded in the drill at 33.6 kg·ha^{-1} in May during planting. The liquid formulation (Furadan 480F) is applied by ground spray equipment on an as-needed basis at 0.6–1.2 L·ha^{-1}. Normally, no more than one application would be made per crop (Monenco Ltd., 1984).

Table 2. Physical and Chemical Properties of Carbofuran

Chemical formula:	$C_{12}H_{15}NO_3$
Molecular weight:	221.26
Elemental analysis:	C, 65.14%; H, 6.83%; N, 6.33%; O, 21.69%
Physical state:	white, crystalline solid
Odour:	odourless, slightly phenolic
Flammability:	not flammable; will support combustion if ignited
Explosive hazard:	none
Corrosive action:	none
Melting point:	153°C to 154°C
Density:	1.180 (20°C)
Vapour pressure:	2×10^{-5} mm Hg (33°C); 1.1×10^{-4} mm Hg (50°C)
Soil adsorption coefficient:	17–160 (depending on soil types)
Octanol/water partition coefficient:	42.45
Solubility (% weight/weight, 25°C):	
Acetone	15
Acetonitrile	14
Benzene	4
Cyclohexanone	9
Dimethyl formamide	27
Dimethyl sulfoxide	25
Ethanol	4
Kerosene	1
N-methyl-2 pyrrolidone	30
Methyl chloride	12
Petroleum ether	1
Xylene	1
Water	0.07 (700 ppm)
Base hydrolysis rate constants:	
5°C	11.4 ± 0.2 L·min^{-1}·mol^{-1}
10°C	18.0 ± 0.3 L·min^{-1}·mol^{-1}
15°C	30.6 ± 0.6 L·min^{-1}·mol^{-1}
25°C	67.0 ± 0.4 L·min^{-1}·mol^{-1}
35°C	163 ± 1 L·min^{-1}·mol^{-1}

Note: Rate constant for 3-hydroxycarbofuran at 15°C is 119 L·min^{-1}·mol^{-1}

Source: Adapted from Life Systems, Inc., 1985, with additional data from McCall et al., 1980; Bowman and Sans, 1983; Kenaga, 1980; and Lemley et al., 1984.

Chemical Characteristics

The physical and chemical properties of carbofuran are presented in Table 2. In addition to the generally reported water solubility of 700 mg·L^{-1} at 25°C, a solubility of 320 mg·L^{-1} at 19°C has also been reported (Life Systems, Inc., 1985). Kenaga (1980) reported a water solubility of 415 mg·L^{-1} (no temperature given).

Carbofuran in the Environment

Carbofuran has the potential to enter the aquatic environment from direct spraying or broadcast of granular formulations, drift deposition of sprayable formulations, and in runoff water from treated fields. Redeposition from the atmosphere is another route of entry, as recent studies have shown the presence of carbofuran in rainwater.

Actual field experiments conducted in southern Alberta, where two ponds were sprayed directly with carbofuran (4.8% flowable) at the recommended rate for grasshopper control of 0.14 kg-ai·ha^{-1}, resulted in detectable residues of 4.2 μg·L^{-1} (surface sample) and 0.4 μg·L^{-1} (bottom sample) in the water of one of the two ponds immediately after spraying (Erickson et al., 1977). Carbofuran could

be detected in water samples from the second pond only at the surface (0.75 $\mu g \cdot L^{-1}$) immediately after spraying. The surface concentration of 4.2 $\mu g \cdot L^{-1}$ declined to 0.3 $\mu g \cdot L^{-1}$ in approximately 7.5 h after the spray application and was below detection limits (<0.1 $\mu g \cdot L^{-1}$) thereafter. The bottom concentration of 0.4 $\mu g \cdot L^{-1}$ declined to 0.1 $\mu g \cdot L^{-1}$ after approximately 2 h and was below detection limits thereafter. The surface concentration of 0.75 $\mu g \cdot L^{-1}$ in the second pond declined to below detection limits after 3.5 h.

Two additional ponds adjacent to fields receiving the 0.14-kg-ai-ha^{-1} application were not directly sprayed. Carbofuran was detected in only one of these ponds as the result of drift from spray applications in the target area. The detection of carbofuran in this pond (0.5 $\mu g \cdot L^{-1}$), however, occurred in samples collected 4.5 h after the spraying of the target area rather than in samples collected immediately after spraying. Because of the high alkalinity of this pond (i.e., 1267–1285 mg $CaCO_3 \cdot L^{-1}$), the routine acidification of samples did not lower the pH sufficiently (i.e., pH 2–4) for the stabilization of the carbofuran for chemical analysis. It can be speculated that some carbofuran was lost from the water samples (by hydrolysis) during shipment to the laboratory, accounting for the lack of detectable carbofuran immediately after spraying (Erickson et al., 1977).

The amount of carbofuran loss from treated fields in runoff water is the result of such interacting factors as target of application (i.e., soil or foliage), timing and intensity of the rainfall after application, formulation and method of the application (granular broadcast, granular seed, furrow incorporation, or spray and percent active ingredient of each), hydrologic characteristics of the treated area, and the chemical and physical characteristics of the soil.

Wauchope and Leonard (1980) attempted to model pesticide runoff mathematically for a variety of pesticides. Their model was based on the physical-chemical properties of the pesticide, the location of the pesticide after application (i.e., foliage, soil surface, or subsurface), the amount of pesticide applied on an area basis, and the dissipation of the pesticide prior to runoff. Using the data from Caro et al. (1973), the model predicted a bulk concentration of 5.9 $mg \cdot L^{-1}$ carbofuran in the runoff from a field treated with 5.41 kg-ai-ha^{-1} as a granular formulation, 2 d after application and 0.12 cm rainfall. The concentration actually observed by Caro et al. (1973) was 1.4 $mg \cdot L^{-1}$. Given the complex nature of pesticide runoff, the model was anticipated by giving only order of magnitude responses. Caro et al. (1973) also observed carbofuran at 1.0 $mg \cdot L^{-1}$ in runoff from a field treated with granular carbofuran at 4.16 kg-ai-ha^{-1}, 2 d after application and 0.04 cm rainfall.

Of the estimated 1092 kg of carbofuran used in 11 southern Ontario agricultural watersheds in 1976, losses to streams draining these areas were calculated to have a mean of 1.5 $mg \cdot ha^{-1} \cdot a^{-1}$ for the period 1976–77. Losses of carbofuran to streams in the same area for the period 1975–76 were determined to be zero (Frank et al., 1982).

In a recent publication, Richards et al. (1987) documented the presence of carbofuran residues in the <0.1–0.5-$\mu g \cdot L^{-1}$ range in rainwater collected from the northeastern United States in 1985. A seasonal pattern of occurrence was reported, as the residues were found only in the spring and summer months.

Krawchuk and Webster (1987) detected carbofuran residues in 8 of 14 ground-water samples from a farm located southwest of Portage la Prairie, Manitoba. The residue concentrations ranged from 11.5 to 158.5 $\mu g \cdot L^{-1}$ in 1982 and from <0.5 to 1.0 $\mu g \cdot L^{-1}$ in 1983. Carbofuran phenol was also identified, and confirmed by mass spectrum analysis, in 5 of the 14 water samples.

Along with published reports specifically concerned with carbofuran in aquatic ecosystem components, surveys of contaminant groups in the aquatic environment, including carbofuran, are also available. These reports and surveys are summarized in Appendix A.

Persistence and Degradation

The persistence of carbofuran dissolved in water is controlled by chemical and biological degradation. These processes, alone or in combination, were responsible for the disappearance of 5.0 $mg \cdot L^{-1}$ carbofuran from natural marsh water, distilled water, sterilized natural marsh water, and sterilized distilled water (Sharom et al., 1980). The similar degradation rates for carbofuran observed under these sterile and nonsterile conditions led to the conclusion that degradation may be primarily chemical.

Hydrolysis is probably the most important chemical reaction aiding in the dissipation of carbofuran. This process is base catalyzed and directly influenced by pH. The half-life of carbofuran in water due to hydrolysis alone varies from approximately 0.2 d at pH 9.5 to 1700 d at pH 5.2. Temperature also has a major influence on the rate of carbofuran hydrolysis with a reported 35% increase in hydrolysis rate for each one degree centigrade temperature increase at ambient temperature. Carbofuran degradation products have even higher rates of hydrolysis than the parent compound and are highly unstable at pH values of approximately 9.5 and temperatures of 37°C–38°C (NRCC, 1979).

Microbial activity is also important in the dissipation of carbofuran in soils. Although microbial communities in the water column are much less dense than those in soil, they have a similar capability for carbofuran degradation. The data reported on the dissipation of carbofuran in water resulting from the combined effects of chemical hydrolysis and microbial degradation generally show rapid reductions in carbofuran concentration. Greenhouse model systems containing only water and five green sunfish (*Lepomis cyanellus* collected from a local pond) demonstrated that carbofuran was reduced from 0.081 to 0.011 $mg \cdot L^{-1}$ in 21 d (Metcalf et al., 1974). These model systems were extremely simple (i.e., fish and water only) and lacked the normal microbial and planktonic assemblages found in surface waters as well as sediment. Thus, it may not be surprising that carbofuran persisted for longer than 21 d in such systems.

By contrast, carbofuran disappearance from rice paddy water treated with a granular formulation (Furadan 2G) at 2 $kg\text{-}ai\text{-}ha^{-1}$ was much faster. The specific formulation used was designed for greater than 90% release within 24 h in periodically agitated water. One and five days after treatment, some of the paddy water was pumped into stagnant ponds where dissipation was monitored (see Table 3). Half-lives of carbofuran in these systems ranged from 48 to 67 h (Seiber et al., 1978).

Additional studies of carbofuran in rice paddy water demonstrated that the chemical hydrolysis of carbofuran to carbofuran phenol occurs rapidly (about 5 d at application rates of 2 $kg\text{-}ai\text{-}ha^{-1}$). The sterilization of the paddy water did not inhibit the degradation of carbofuran, but did produce a buildup of carbofuran phenol, which was not observed in the unsterilized water. Thus, while the degradation of carbofuran to carbofuran phenol is apparently due to chemical hydrolysis, the removal of carbofuran phenol is substantially accelerated by the presence of microbes (Siddaramappa et al., 1978; Siddaramappa and Seiber, 1979).

Table 3. Loss of Carbofuran from Paddy and Pond Water in the Field

Paddy		Pond A		Pond B	
Elapsed time (d)	Conc. ($mg \cdot L^{-1}$)	Elapsed time (d)	Conc. ($mg \cdot L^{-1}$)	Elapsed time (d)	Conc. ($mg \cdot L^{-1}$)
1	2.0	0.80	1.8	1	0.45
2	1.6	1.2	1.5	2	0.38
3	1.3	4	0.59	3	0.34
5	0.76	5	0.22	4	0.14
6	0.68	6	0.18	8	0.01
8	0.34	7	0.06		
13	0.02	8	0.02		
$K_{ob}(h^{-1})$*	1.0×10^{-2}		1.4×10^{-2}		1.2×10^{-2}
$t^1/_2$ (h)*	67		48		55

Source: Seiber et al., 1978.

Note: Average pH's at 8:00 a.m. and 5:00 p.m. were 7.8 and 8.5, respectively; average temperatures at 8:00 a.m. and 5:00 p.m. were 26°C and 30°C, respectively.

Maximum concentration of carbofuran in paddy water was 2.3 $mg \cdot L^{-1}$.

K_{ob} = observed rate constants

$t^1/_2$ = calculated half-lives

*Calculated for days 1-8 (paddy), days 0-4 (pond A), and days 0-4 (pond B).

Similar studies of degradation in natural and sterilized paddy water by Deuel et al. (1979) also confirmed the nonbiological dissipation of carbofuran. In this latter case, however, the paddy water pH ranged between 6.0 and 6.5. It has been speculated that the removal of carbofuran can also result from clay-surface catalyzed hydrolysis. Carbofuran applied at a rate of 0.56 kg-ai-ha^{-1} (as a broadcast of an unspecified granular formulation) to experimental 300-m^2 plots was degraded to <0.02 kg-ai-ha^{-1} in 96 h during a 1973 field trial.

Carbofuran degradation in natural ponds (pH about 8.5) following direct aerial application (0.14 kg-ai-ha^{-1}) showed a maximum carbofuran persistence from 10 to 21 h (Erickson et al., 1977). Degradation was reported to be the direct result of hydrolysis occurring at the carbamate linkage yielding carbofuran phenol as the main degradation product. No information was given regarding the possibility of microbial degradation. Concurrent laboratory studies demonstrated a 70% or greater hydrolysis of carbofuran in the natural waters at the end of 30 d. Natural pond water adjusted to pH 2.5 and 5.0 did not produce hydrolytic degradation of carbofuran, whereas increasing the pH to 12.5 resulted in the immediate and complete hydrolysis of carbofuran. Natural waters of pH 9.5 and 8.25 showed dramatic (i.e., 45%–60%) reductions of carbofuran within the first 10 d. Variations in hydrolytic degradation among the natural waters can apparently be affected by variations in the natural salts producing a buffering effect and slowing hydrolysis (Erickson et al., 1977).

Additional published studies concerning carbofuran degradation in water under Canadian conditions were not found. Other relevant data may exist, but are not published at this time. An example is the pesticide sampling and analysis program conducted by Alberta Environment in the southern part of the province (Eco/Log Week, 1986). The sampling and analysis program reportedly found 5 μg-L^{-1} in irrigation water supplies.

In their study of the persistence of carbofuran and its metabolites in rice paddy water, Deuel et al. (1979) used laboratory experiments to determine if photodecomposition and/or volatilization contributed to the dissipation of the insecticide. Significant photodecomposition has been observed in deionized water irradiated with natural sunlight for 96 h. While the potential for substantial photodecomposition may exist, field studies are needed to fully quantify the significance of this degradative process. Volatilization of carbofuran and 3-ketocarbofuran from water in the laboratory was found to be insignificant (Deuel et al., 1979).

One mechanism of carbofuran dissipation from rice paddies that has received limited attention is volatilization and exudation of the parent compound and its metabolites through the leaf surfaces of the rice plants. This mechanism, whereby carbofuran is taken up by the plant roots and translocated to the rapidly transpiring and fully developed leaves, allows the loss of the pesticide by direct volatilization through the stomata or by volatilization from or removal of the guttation fluid present on plant leaves. The quantity of carbofuran removed by this mechanism depends on the manner in which the pesticide is applied (i.e., broadcast to paddy water or soil or soil incorporation) and the various meteorological conditions to which the plants are exposed. The laboratory studies investigating this mechanism have generally revealed carbofuran losses via this route of less than 10% of the applied pesticide. Field conditions might produce higher losses (Siddaramappa and Watanabe, 1979; Ferreira and Seiber, 1981).

Information is scarce concerning the environmental fate and persistence of carbofuran in true lacustrine sediments. Carbofuran has been found in sediments of farm ponds deliberately treated with the chemical (Klaassen and Kadoum, 1979), but no attempt was made to examine the various processes in the sediment responsible for carbofuran dissipation. The available information concerning carbofuran adsorption on selected soils, its solubility in water, and soil-water equilibration indicates that sediment concentrations of carbofuran are not anticipated to be substantially higher than the associated water concentrations (NRCC, 1979).

Information is available concerning the fate and persistence of carbofuran in flooded soils used for rice cultivation. In neutral and alkaline flooded soils, degradation of carbofuran is accomplished by both chemical and biological mechanisms. Some acid soils are also capable of attaining near-neutral pH after flooding (Sethunathan et al., 1982; Venkateswarlu and Sethunathan, 1984).

The degradation of carbofuran in flooded soil is essentially a hydrolytic reaction producing carbofuran phenol, although Venkateswarlu and Sethunathan (1978) believe that this chemical hydrolysis may be catalyzed or mediated by the microflora of the flooded soil. Anaerobic conditions

in flooded soils apparently enhance the hydrolysis of carbofuran to carbofuran phenol and 3-hydroxycarbofuran. Further degradation of these metabolites to carbon dioxide and water requires aerobic conditions and is primarily accomplished by the microbial community.

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The maximum acceptable concentration (MAC) of carbofuran listed in the Guidelines for Canadian Drinking Water Quality, 1987 is 90 $\mu g \cdot L^{-1}$ (Health and Welfare Canada, 1987). This value is based on a no-observed-effect level (NOEL) of 0.01 $mg \cdot kg^{-1} \cdot d^{-1}$ from a 2-year rat-feeding study in which cholinesterase depression and body weight loss occurred at higher doses.

Freshwater Aquatic Life

Levels and Fate in the Aquatic Environment

The fate of carbofuran in aquatic ecosystems has been studied in both field and laboratory settings. The distribution and retention of carbofuran (applied as Furadan 4 Flowable, 43.8% ai) in farm ponds were examined by Klaassen and Kadoum (1979). Carbofuran was applied for 2 years to two farm ponds in northeast Kansas to obtain concentrations in the water of 0.025 $mg \cdot L^{-1}$ (first year) and 0.05 $mg \cdot L^{-1}$ (second year). The treated ponds and a control pond were sampled 1–6 d before being treated, within 3 d after treatment, about 3 weeks after treatment, and twice more before winter. Samples from each pond consisted of water, sediment, and biological components (i.e., fish, tadpoles, crayfish, and zooplankton).

During the first year (initial carbofuran concentration 0.025 $mg \cdot L^{-1}$), only the surface water sample from one pond collected 1 d after treatment contained detectable concentrations of carbofuran (i.e., 0.0106 $mg \cdot L^{-1}$). Carbofuran in the remaining samples [i.e., sediments from shallow and deep areas, zooplankton, small- and medium-sized black bullheads (*Ictalurus melas*), and small, medium, and large bluegill (*Lepomis macrochirus*)] was below the detection limit of 0.0004 $mg \cdot kg^{-1}$. All samples collected 26, 50, and 85 d after treatment did not contain detectable levels of carbofuran. The pH of the water at the time of sampling was not given, but over the 2-year study period, the pH of the pond water was reported to have ranged from 6.6 to 9.7 (surface) and from 6.8 to 8.5 (bottom).

Three days after similar treatment of a second pond, carbofuran was detected only in the surface water (0.0054 $mg \cdot L^{-1}$) and in the sediment from the shallow portion of the pond (0.044 $mg \cdot kg^{-1}$). At 21 and 77 d, carbofuran could not be detected in any samples (Klaassen and Kadoum, 1979). The pH of the water at the time of sampling was not given, but over the 2-year study period, the pH of the pond water was reported to have ranged from 7.7 to 8.3 (surface) and from 7.4 to 8.0 (bottom).

During the second year, carbofuran (initial concentration 0.05 $mg \cdot L^{-1}$) was found in the surface water (0.015 $mg \cdot L^{-1}$), in shallow water sediment (0.0264 $mg \cdot kg^{-1}$), and in deep water sediment (0.0462 $mg \cdot kg^{-1}$) 3 d after treatment. Carbofuran was not detected in any of the remaining samples (i.e., zooplankton and fish). Likewise, carbofuran was not detected in any sample collected 25, 62, and 95 d after treatment.

A second pond treated with atrazine (initial concentration 0.3 $mg \cdot L^{-1}$) and carbofuran (initial concentration 0.05 $mg \cdot L^{-1}$) during the second year had carbofuran detected in the surface water 2 d (0.0335 $mg \cdot L^{-1}$) and 23 d (0.0015 $mg \cdot L^{-1}$) after treatment. Carbofuran was detected only in shallow water sediment (0.0595 $mg \cdot kg^{-1}$) 2 d after treatment. All remaining samples collected 2, 23, 54, and 92 d after treatment did not contain carbofuran at detectable levels (detection limit 0.4 $\mu g \cdot L^{-1}$).

Carbofuran degraded quite rapidly in these farm ponds and was not accumulated or bioconcentrated by the aquatic fauna (Klaassen and Kadoum, 1979).

Isensee and Tayaputch (1986) used rice paddy laboratory microcosms to study the fate and behaviour of radiolabelled carbofuran and the potential effects of carbofuran residues on the fish *Gambusia affinis*. Carbofuran was added as a 1500-g layer of soil treated with carbon-14 ring-labelled carbofuran (purity greater than 97%, 39.4 mCu/mmole specific activity) at concentrations of 6 and 12 $mg \cdot kg^{-1}$. The treated soil layer was approximately 1 cm in depth and covered 5 kg of untreated soil containing rice seeds. Three replicates of each concentration plus two controls had soil moisture maintained at approximately 24% (w/w) for 18 d.

After that period, 14 L of water were added to each microcosm, flooding the soil to a depth of approximately 6 cm. Fish were added to the microcosms the next day. Soil samples were collected 4 d prior to flooding and 7, 14, 28, and 42 d after flooding. Water samples were taken at 2-d intervals for total radioactive carbon. Additional water samples were taken at weekly intervals for recovery of carbon-14 carbofuran plus metabolites. Fish were sampled 1, 3, 7, 15, and 30 d after they were added to the microcosms. The fish remaining after 30 d were placed in carbofuran-free water and sampled after 4 and 10 d.

The radioactive carbon in water reached a maximum concentration of 12.2%–13.1% of the total carbon-14 applied to the soil at the beginning of the experiment. This maximum in the radioactivity appeared 9 d after flooding and then decreased to 4.1%–4.6% by day 44 for the 6–12-mg·kg^{-1} treatments.

Results of the analysis of fish in the microcosms were of limited value due to unexplained mortality in some of the treatment and control microcosms. The extractable ^{14}C in the fish on day 3, however, accounted for about 60% of the total ^{14}C in fish (Isensee and Tayaputch, 1986). This extractable ^{14}C decreased to 10% by day 30, apparently indicating that the ^{14}C was being incorporated into fish tissue. There was no mention of the possibility of loss due to excretion. The total ^{14}C concentrations in the fish increased continuously with time to day 30 with little loss of the radiocarbon after placement in carbofuran-free water for 11 d. The maximum carbofuran concentration in the fish extracts was 88 ng·g^{-1} (for the 12-mg·kg^{-1} treatment) and occurred 1 d after the fish were introduced into the microcosm. In this study, carbofuran accounted for 5%–14% of the total ^{14}C in the fish extracts. The identity of the unextractable ^{14}C was unknown. Rice seedlings were not analyzed in this experiment.

Two weeks after the radiolabelled ^{14}C-carbofuran-contaminated soil application (and prior to flooding), about 50% of the radiocarbon had leached into the untreated soil. Seven days after flooding, less than 20% of the radiocarbon remained in the treated soil. The extractable radiocarbon from both the treated surficial and untreated subsurface soil decreased with time and corresponded to an increase in unextractable ^{14}C-activity. Soil adsorption of the radiocarbon carbofuran or its labelled products probably account for the increase in unextractable ^{14}C-activity.

A soil extract taken at day 14 (4 d prior to flooding) contained 70%–76% of the recovered ^{14}C-activity as carbofuran. Ten percent to 14% of the recovered ^{14}C-activity was identified as 3-ketocarbofuran. After flooding, carbofuran accounted for 85%–90% of the recovered ^{14}C-activity. No 3-ketocarbofuran was detected, however, and no other metabolites were found. Rapid degradation of the 3-ketocarbofuran may have accounted for its disappearance after 7 d or more of flooding and the onset of anaerobic conditions.

Johnson (1986) used multicomponent aquatic microcosms simulating a northern prairie wetland to assess the effects of 0.01-, 0.1-, and 1.0-mg·L^{-1} carbofuran concentrations (technical grade, 99% ai). Sediment and topsoil from a permanent wetland area and adjacent agricultural field (previously analyzed and determined to be carbofuran-free) were used for the soil substrate in the microcosms. Microcosm water was a mixture of standard reconstituted water and natural wetland water at an 8:1 v/v ratio. Although the chemical parameters of the resulting mixture were not given, the reconstituted water had a pH of 7.2 and a total hardness of approximately 60 mg·L^{-1}. Approximately 1 week was allowed for the stabilization of water, suspended solids, and substrate before three types of aquatic plants were introduced (i.e., surface-floating *Lemna*, nonrooting, submergent *Ceratophylum*, and rooting submergent *Elodea*). Natural communities of invertebrates and algae developed within each microcosm within the 2-week period between aquatic plant establishment and carbofuran treatments of 0.01, 0.1, and 1.0 mg·L^{-1} as introduced into the water. In addition, 25 mature, gravid daphnids from a healthy reproducing culture were introduced into each microcosm unit about 48 h prior to carbofuran treatment. These populations were then monitored in each microcosm.

In the 0.01- and 0.1-mg·L^{-1} treatments, viable populations of *Daphnia magna* were still established after 1 d exposure. Similar population development required 4 d in the 1.0-mg·L^{-1} treatment. Thereafter, the survival rates of the adults and instars and the number of gravid females were within control values for all three treatments (Johnson, 1986).

The effect of carbofuran treatments on phytoplankton was assessed after 30 d in the microcosms by measuring the growth inhibition of the water to the green alga *Selenastrum capricornutum*. The carbofuran-treated water at 0.01 $mg \cdot L^{-1}$ showed no effects, whereas the 0.1- and 1.0-$mg \cdot L^{-1}$ treatments produced a mild stimulation of algal growth (Johnson, 1986).

Gross primary production, community respiration, and macrophytic biomass in the microcosms were not influenced by carbofuran treatments. As well, water pH, alkalinity conductivity, total nitrogen, total phosphorous, and total organic carbon were not affected. In addition, respiratory electron transport system activity, glucose metabolism, oxygen consumption, and alkaline phosphatase activity in the microcosm hydrosoils were not changed by the carbofuran treatments (Johnson, 1986).

An agro-microcosm designed by Koeppe and Lichtenstein (1982) allowed an investigation of the movement of radiolabelled carbofuran from soils by percolating water into an aquatic environment consisting of lake water, lake sediment, aquatic plants (*Elodea*), and fish (*Poecilia*). After 3 weeks, the aquatic microcosm contained approximately half of the carbofuran added by percolated water. The other half was apparently lost by degradation. Approximately 75% of the radiocarbon in the aquatic microcosm was contained in the lake sediment, much of it unextractable. In contrast to the 49% removal of carbofuran by percolating water, unpercolated soils contained 80% of the applied carbofuran after 3 weeks. Carbofuran was the major compound recovered from control (i.e., unpercolated) and percolated soils. The metabolites identified in the soils were 3-ketocarbofuran and 3-hydroxycarbofuran (Eisler, 1985).

The microcosms used by Yu et al. (1974) contained seedling sorghum plants (*Sorghum halopense*), salt-marsh caterpillars (*Estigmene acrea*), and a variety of aquatic organisms [e.g., unidentified frogs, fish, snails, mosquito larvae, a filamentous alga (*Oedogonium cardiacum*), clams (*Corbicula manilensis*), crabs (*Uca minax*), zooplankton (*Daphnia*), and an aquatic vascular plant (*Elodea*)]. Carbofuran was introduced into four of these microcosms as 5 mg radiolabelled carbofuran in 0.5 mL of acetone applied to the leaves of the sorghum plants. This was equivalent to an application rate of 0.454–$kg \cdot ai \cdot ha^{-1}$. The carbofuran introduced into two of the four microcosms was labelled only on the carbofuran ring, whereas the other two microcosms received carbofuran labelled only on the carbonyl or methylcarbamate group.

The quantity of carbofuran introduced into these microcosms was sufficient to kill most of the organisms and thus a continual restocking of organisms every 5–7 days was necessary. Organisms stocked 20 d after the introduction of the carbofuran survived and were sacrificed for radiocarbon analysis after a 10-d exposure.

In the microcosms containing the ring–labelled carbofuran, the highest radioactivity (i.e., highest concentration of carbofuran metabolite) appeared in the plants (*O. cardiacum* and *Elodea*) and the invertebrates. Water and fish contained carbofuran metabolites at concentrations generally an order of magnitude below those in the other aquatic components. This same pattern was observed in the tanks receiving the carbonyl-labelled moiety except for the differences in concentrations of carbofuran metabolites in the water, which were two orders of magnitude below the concentration in the fish and three orders of magnitude below the concentration in the invertebrates and aquatic plants. The differences between the amount of radioactivity in the tanks receiving ring- and carbonyl-labelled carbofuran indicated that carbofuran was hydrolyzed to carbofuran phenol and N-methylcarbamic acid. The parent compound (i.e., unmetabolized carbofuran) was not found in any of the last stock of organisms. Identification of specific metabolites was limited due to the separation techniques used (thin-layer chromatography) and the availability of standards. Carbofuran was found in the unfiltered water (0.0015 $mg \cdot L^{-1}$) after treatment, but it was unclear from the presentation of results whether the carbofuran was actually dissolved or was adsorbed to suspended particulate material (Yu et al., 1974).

This microcosm study demonstrated that an application rate of 0.454–$kg \cdot ai \cdot ha^{-1}$, even if applied only to the leaves of terrestrial plants, could be highly toxic to adjacent aquatic organisms. Even after 20 d, when the concentration of the applied carbofuran had decreased below acutely toxic levels, both the parent compound and its metabolites were still present in sufficient quantity in the water for uptake by aquatic organisms, especially invertebrates (Yu et al., 1974).

Accumulation and Elimination of Carbofuran in Aquatic Biota

The studies of Jash and Bhattacharya (1983), Bhattacharya (1985a, 1985b), and Bakthavathsalam and Reddy (1983) demonstrated or implied that carbofuran taken up by fish when exposed to sublethal concentrations can be depurated from the organism after several days in carbofuran-free water. Whether or not the depuration consisted of carbofuran or a metabolic product of carbofuran was not discussed by these authors.

Similarly, Gill (1980) suggests that the hydroxylation of carbon ring structures (such as the production of 3-hydroxycarbofuran) generally increases the solubility of these types of molecules and aids in their elimination from the organism.

Metcalf et al. (1974) examined the uptake of carbofuran in green sunfish (*Lepomis cyanellus*) from water containing 0.1 $mg \cdot L^{-1}$. After 24 d at 13°C–20°C, less than 10^{-5} $mg \cdot L^{-1}$ carbofuran was found in the whole organism extracts. Metcalf et al. (1971) demonstrated that the mosquito fish (*Gambusia affinis*) did not accumulate carbofuran from the water of microcosms previously treated with the insecticide.

In an effort to quantify the relationship between pesticide water solubility and the bioconcentration of pesticides by caddisfly (*Triaenodes tardus*) eggs, Belluck and Felsot (1981) found exposure to 8 $mg \cdot L^{-1}$ did not result in bioconcentration.

Significant bioconcentration of carbofuran by aquatic organisms in excess of the concentration in the surrounding water has not been found. The physical-chemical characteristics of the compound, as expressed by the octanol-water partition coefficient (K_{ow}), supports this finding. The K_{ow}s for carbofuran have been reported to be 17, 42.5, 40.0, and 210 (Graham, 1977; Bowman and Sans, 1983).

The value of 42.5 is from an actual measurement of the partitioning of carbofuran between n-octanol and distilled water. Other K_{ow}s reported from the literature are estimates based on other physical or chemical characteristics of the compound. Using the K_{ow} and the relationship between K_{ow} and compound bioconcentration developed by Neely et al. (1974), a bioconcentration factor of 10 is derived. Other bioconcentration factors of 2.5, 5, and 6 have also been reported (NRCC, 1979). These bioconcentration factors (i.e., tissue concentration/water concentration) are very low and have been substantiated in laboratory tests.

In fish uptake and depuration experiments, most of the carbofuran (as metabolites) was concentrated in the viscera after equilibrium was reached. After exposure stopped, approximately 97% of the carbofuran metabolites were eliminated in 3 d. A depuration half-life of 4 d (after equilibrium exposure to 66 $\mu g \cdot L^{-1}$ carbofuran) has been reported (NRCC, 1979).

Acute Toxicity to Aquatic Organisms

Fish

Two references (Mauck, 1972; Carter and Graves, 1973) cited in NRCC (1979) support its contention that fish species may be adversely affected when exposed to carbofuran at levels as low as 0.08 $mg \cdot L^{-1}$ for short periods. Since publication of that document, many additional acute toxicity tests have been conducted using carbofuran. A summary of these tests is presented in Appendix B. The additional toxicity data from these tests support the NRCC document's implication that 0.08 $mg \cdot L^{-1}$ is at the lower end of toxic concentrations for fish exposed for periods of less than 96 h (NRCC, 1979).

Sublethal symptoms in fish exposed to acutely toxic concentrations of carbofuran can include hypoactivity, body paralysis, lateral curvature of the spine (usually with localized hemorrhaging), loss of equilibrium, and opercular and mouth paralysis.

Lethality in several species of fish in Texas rice fields reported by NRCC (1979) after the application of carbofuran as 3% granules (0.56 $kg \cdot ai \cdot ha^{-1}$) cannot be directly attributed to carbofuran due to the use of another insecticide for seed treatment. By contrast, an application rate of 0.67 $kg \cdot ai \cdot ha^{-1}$ of carbofuran alone killed less than 10% of green sunfish (*Lepomis cyanellus*) and mosquito fish (*Gambusia affinis*) in flooded rice plots (Davey et al. 1976). Although formal 96-h LC_{50} values were not calculated, Bhattacharya (1985b) reported that 0.313, 0.375, and 0.462 $mg \cdot L^{-1}$ carbofuran caused 0%, 10%, and 20% mortality, respectively, in the fish *Channa punctatus*. The same percentage mortality in the fish *Anabas testudineus* was produced by 0.150, 0.300, and 0.450 $mg \cdot L^{-1}$ carbofuran.

The entries in Appendix B under "test conditions" indicate that with the exception of Hansen and Parrish's (1977) study with sheepshead minnows (*Cyprinodon variegatus*), acute toxicity testing with carbofuran has been conducted without a measurement of actual carbofuran concentrations in the test chambers.

Invertebrates

Short-term carbofuran toxicity to invertebrates (Appendix B) has received much less attention than acute toxicity to fish. Eisler (1985) implied that benthic invertebrates (e.g., worms) were the most resistent aquatic species and fish were the most sensitive. The data of Hartman and Martin (1985), Johnson (1986), and Karnak and Collins (1974), however, seem to indicate that invertebrates (both planktonic and benthic) may be very sensitive to carbofuran. In general, the 48-h LC_{50}s for *Daphnia pulex* (Hartman and Martin, 1985) and the 48-h LC_{50}s for *Daphnia magna* and *Chironomus riparius* (Johnson, 1986) are lower than most of the 96-h acute toxicity values for fish species (see Appendix B).

Acute toxicity tests were used to estimate a "harmless" concentration of Furadan 3G (active ingredient 3% carbofuran) of 4.4 and 3.2 $mg \cdot L^{-1}$ for the oligochaete worms *Tubifex tubifex* and *Limnodrilus hoffmeisteri*, respectively (Dad et al., 1982).

Observations of the behaviour and oxygen consumption of adult freshwater prawns (*Macrobrachium kistnensis*) exposed to 50, 150, and 300 $\mu g \cdot L^{-1}$ carbofuran were made by Pawar and Katdare (1984). Exposure to 50 $\mu g \cdot L^{-1}$ caused significant increases in oxygen consumption after 2 h followed by significant decreases in oxygen consumption at 12, 24, 48, and 96 h. Behaviour during the exposure period was reported as excitation for the first 4 h with subsequent normal behaviour thereafter. The higher concentrations caused spontaneous movements, hyperactivity, loss of balance, and finally paralysis. The behaviour was accompanied by immediate and significant increases in oxygen consumption followed by significant decreases prior to death.

Static exposures of groups of 40 crawfish (*Procambarus clarkii*) per test to 0.22, 0.44, 0.88, and 2.64 $mg \cdot L^{-1}$ Furadan (formulation and percent active ingredient not given) produced 33%, 55%, 75%, and 78% mortality, respectively (Ekanem et al., 1981).

Plants

Acute toxicity tests with duckweed (*Lemna minor*) and 10 $mg \cdot L^{-1}$ carbofuran showed no effects on growth. Likewise, 10 $mg \cdot L^{-1}$ carbofuran had no effect on sprouting or early growth of sago pondweed (*Potamogeton pectinatus*) tubers (Hartman and Martin, 1985).

Sublethal Reactions and Chronic Toxicity in Aquatic Organisms

Fish

Kulshrestha et al. (1986) exposed fingerlings of three Indian major carps (*Labeo rohita*, *Catla catla*, and *Cirrhinus mrigala*) to Furadan (carbofuran 3G) for 30 d. Unfortunately, the data derived from this experiment were only presented graphically and not discussed in the text of the article. From the graphic presentation of the data, it appears that 4.0 $mg \cdot L^{-1}$ Furadan did not cause mortalities during the 30-d exposure period. Increasing concentrations of Furadan caused increasing mortalities among the test populations in an almost linear fashion. The maximum concentration used (8.5 $mg \cdot L^{-1}$ carbofuran) caused from 20% mortality in *L. rohita* to 30% mortality in *C. mrigala*. Ninety-six-hour LC_{50} values for the three species ranged between 4.7 and 5.1 $mg \cdot L^{-1}$ carbofuran. The estimated maximum acceptable toxicant concentration for *L. rohita*, *C. catla*, and *C. mrigala* was determined to be in the ranges of 2.7–3.3, 2.7–3.3, and 2.4–3.0 $mg \cdot L^{-1}$, respectively.

Kulshrestha and Arora (1986) studied the effect of carbofuran on egg mortality and hatching in *Cyprinus carpio*. The carbofuran formulation used (i.e., 3G) caused 68% mortality in eggs after 60- to 72-h exposure to 0.2 $mg \cdot L^{-1}$. The 0.1-$mg \cdot L^{-1}$ concentration caused slightly less egg mortality (approximately 55%) for the same exposure time. Advancement in hatching time did not seem to be significantly affected relative to the control.

Symptoms of apparent stress or mortality were not observed when the air-breathing catfish *Clarias batrachus* was exposed to 0.5 $mg \cdot L^{-1}$ carbofuran for 30 d (Sadhu and Mukhopadhyay, 1985). Upon histological examination of the testes, however, distinct morphological damage (i.e., seminiferous tubule wall disintegration) was observed. As well,

spermatids in the seminiferous tubules appeared clumped in the carbofuran-exposed fish. The testes also appeared to accumulate carbofuran to a slight extent. The significance of this, however, was not discussed by the authors.

Hansen and Parrish (1977) examined the effects of carbofuran on the survival of sheepshead minnow (*Cyprinodon variegatus*) parental fish and two life-stages of their progeny during 19-week exposure periods. Carbofuran concentrations of 0.049 and 0.1 $mg \cdot L^{-1}$ caused mortality among the adult population. Hatching success of the progeny of the surviving adults in 0.049 $mg \cdot L^{-1}$ carbofuran was significantly less than the control. As well, mortality of F_1 fry in the population exposed to 0.023 $mg \cdot L^{-1}$ was significantly greater than the control. Growth of the surviving fry in the 0.023- and 0.049-$mg \cdot L^{-1}$ concentrations was the same as the control. Maximum acceptable toxicant concentrations of carbofuran for this fish species were determined to be between 0.015 and 0.023 $mg \cdot L^{-1}$. Verma et al. (1982) computed a presumably safe concentration of 0.0142 $mg \cdot L^{-1}$ using acute toxicity data and an arbitrary application factor of 0.026 for carbamate pesticides in general.

Histopathological effects of 120-h exposure to 0.56 $mg \cdot L^{-1}$ and 6-h exposure to 1.56 $mg \cdot L^{-1}$ Furadan (75% technical grade) in the climbing perch (*Anabas testudineus*) were examined by Bakthavathsalam et al. (1984). The sublethal exposure (i.e., 0.56 $mg \cdot L^{-1}$) produced severe liver cord disarray, destruction of hepatocytes and necrosis, shrinkage of glomeruli and epithelial cells in renal tissue, coalescence of adjacent renal tubules and interepithelial cells, deformation of renal tubules, vacuolization of the intestinal submucosa and circular muscle, and dilation of the intestinal columnar and goblet cells. The 24-h lethal exposure (i.e., 1.56 $mg \cdot L^{-1}$) also exhibited the above histopathological changes in addition to atypical enlarged liver cells, honeycombed renal glomeruli, degeneration of renal epithelial cells, severe degeneration in the brush borders of renal tubules, disorganization in the intestinal serosa, aggregation of intestinal muscle, severe damage to intestinal submucosa, and necrotic mucosal folds in the intestine.

The mode of action of carbofuran is based on its inhibition of acetylcholinesterase activity, and many carbofuran effects on fish are related to this enzyme system. Concentrations of carbofuran as low as 0.19 $mg \cdot L^{-1}$ have been reported to cause a 50% inhibition of brain acetylcholinesterase (AChE) activity in the channel catfish (*Ictalurus punctatus*) (NRCC, 1979). Jash and Bhattacharya (1983) examined the *in vitro* inhibition of brain AChE activity in two freshwater fish (*Channa punctatus* and *Anabas testudineus*) in terms of their previous exposure to carbofuran. In fish previously exposed to carbofuran, the time required for 50% inhibition of AChE (*in vitro*) was found to decrease at the 0.300- to 0.462-$mg \cdot L^{-1}$, inclusive, levels of treatment. The lowest treatment concentration of 0.150 $mg \cdot L^{-1}$ failed to cause a response different from the control populations. This indicated that previous exposure of the test fish resulted in sublethal brain AChE inhibition. Subsequent exposures, prior to complete depuration, enhanced the previous inhibition response. Fish previously exposed to carbofuran (i.e., 0.300 to 0.462 $mg \cdot L^{-1}$) and then allowed to depurate in carbofuran-free water gradually regained the *in vitro* AChE inhibition times characteristic of unexposed fish over a period of 10 to 20 d. At 30 d, however, the time for 50% AChE inhibition (*in vitro*) again decreased below control levels for one species (*C. punctatus*) previously treated with 0.312 and 0.375 $mg \cdot L^{-1}$ carbofuran. This decrease was attributed to a delayed neurotoxic effect in the more sensitive of the two species tested.

Bhattacharya (1985a, 1985b) again described the *in vitro* effect of carbofuran on AChE activity from the brains of two freshwater teleosts previously exposed for 48 h to carbofuran (75% ai) and in untreated controls. The *in vitro* inhibition of AChE from carbofuran-exposed fish indicated an incorporation of carbofuran into the brain, which was gradually reversed with time spent in carbofuran-free water. After 48-h exposure to 312 $\mu g \cdot L^{-1}$ carbofuran and subsequent transfer to carbofuran-free water, a period of 60 d elapsed before brain AChE activity returned to normal levels in *C. punctatus*. The same length of time was required for *A. testudineus* to recover from a 48-h exposure to 150 $\mu g \cdot L^{-1}$ carbofuran.

Exposure to higher concentrations of carbofuran (375 and 462 $\mu g \cdot L^{-1}$ for *C. punctatus* and 300 and 450 $\mu g \cdot L^{-1}$ for *A. testudineus*) revealed a decrease in the time required to attain control level AChE activity after 48-h exposures. *C. punctatus* exposed to 462 $\mu g \cdot L^{-1}$ for 48 h attained control level AChE activity in 10 d. *A. testudineus* exposed to 450 $\mu g \cdot L^{-1}$ for 48 h required 30 d to attain normal AChE activity. The data demonstrated a trend of faster

AChE activity recovery in fish exposed to higher sublethal carbofuran concentrations. However, a delayed neurotoxic effect previously reported by Jash and Bhattacharya (1983) was again observed in *C. punctatus* exposed to 375 and 312 $\mu g \cdot L^{-1}$ carbofuran for 48 h.

Forty-eight-hour exposures to carbofuran (concentration not given) caused a 55%–60% reduction in AChE activity in the Indian Siluroid fish (*Heteroprieustes fossilis*). *In vitro* incubations of fish brain tissue with 0.32 $mg \cdot L^{-1}$ carbofuran caused a 50% reduction in AChE activity in 40 min (Sur and Ghose, 1978).

The impact of carbofuran (as Furadan) on the succinate dehydrogenase activity in brain, intestine, liver, muscle, and kidney tissue of *Anabas testudineus* was studied by Bakthavathsalam and Reddy (1983). Fish exposed to two concentrations of Furadan were examined for succinate dehydrogenase activity. Enzyme activity in the control (unexposed) fish was relatively high and stable, whereas significant sporadic variations occurred in the various exposed fish at least during some portions of the 120-h exposure to 0.56 $mg \cdot L^{-1}$ carbofuran. Fish exposed for 6 h to 1.56 $mg \cdot L^{-1}$ showed elevated (compared to control) levels of enzyme activity in gill, brain, intestine, and liver tissue and decreased enzyme activity in muscle and kidney tissue. The variations in succinate activity observed over the course of carbofuran exposure are speculated to be the result of carbofuran metabolism within individual tissues, carbofuran neurotoxicity within the brain, and depuration processes occurring within individual tissues and throughout the organism as a whole.

Bakthavathsalam and Reddy (1985) examined the effects of 0.56- and 1.56-$mg \cdot L^{-1}$ concentrations of Furadan (75% ai) on aquatic, aerial, and total oxygen consumption by the climbing perch (*Anabas testudineus*). The lowest Furadan concentration (0.56 $mg \cdot L^{-1}$) caused a significant reduction in aerial and total oxygen consumption immediately after exposure. Aquatic oxygen consumption was severely affected in later exposure periods with the test fish depending more on aerial respiration than aquatic respiration to satisfy its oxygen needs. Aerial and aquatic oxygen consumption varied over the different exposure times, but resumed a normal pattern after 96 h. The higher (1.56 $mg \cdot L^{-1}$) concentration of Furadan caused a greater decrease in both aerial and aquatic oxygen consumption within the first hour of exposure than was observed for the lower concentration. Subsequent increases in oxygen consumption to normal and above-normal levels were probably associated with the more irregular and erratic activity of the fish at the higher Furadan concentration.

The effect of carbofuran (apparently 100%, but no formulation information given) on the *in vitro* metal-activated hydrolysis of adenosine triphosphate (ATP) by bluegill (*Lepomis macrochirus*) liver mitochondria was examined by Hiltibran (1982). Carbofuran (1.5 μmol/mL) significantly decreased magnesium-activated hydrolysis of ATP, whereas the same concentration of carbofuran did not affect calcium-activated hydrolysis of ATP. The alteration of magnesium-activated ATP hydrolysis indicated that carbofuran has the potential to interact with various enzyme-metal complexes. Exactly how this interaction occurs is unknown. In fact, the basis for this study was questionable as the physiological roles of magnesium- and calcium-activated ATP hydrolysis are unknown.

Additional studies of the alteration of enzyme-catalyzed reactions in the African catfish (*Mystus vittatus*) were conducted by Verma et al. (1981b). Sublethal concentrations of carbofuran (i.e., 0.062, 0.031, and 0.021 $mg \cdot L^{-1}$) inhibited the activities of acid, alkaline, and glucose-6-phosphatases in liver, kidney, and gill tissue. The authors offered no clear explanation for the observed alterations in enzyme activity, but speculated that the inhibition of acid and alkaline phosphatases might be caused by the uncoupling of oxidative phosphorylation. The inhibition of glucose-6-phosphatase will cause an alteration in glycogen and carbohydrate metabolism in the effected organism. Verma et al. (1981a) also found significantly elevated levels of serum transaminases in *M. vittatus* exposed to 0.031 or 0.062 $mg \cdot L^{-1}$ carbofuran for 30 d.

Bakthavathsalam and Reddy (1981) examined the effect of carbofuran on muscle and liver lipid levels in the climbing perch (*Anabas testudineus*). Furadan (technical grade, 75% purity) at a concentration of 0.56 $mg \cdot L^{-1}$ caused a significant increase in the mean lipid levels of muscle and liver tissue over the 120-h exposure period. The same results were observed at the end of a 6-h exposure to 1.56 $mg \cdot L^{-1}$ carbofuran. The authors speculated that the tissue lipid levels reflected the rates of

carbofuran metabolism. Additional discussion about the meaning of the results was not given.

Mukhopadhyay et al. (1982) examined the effects of 0.5 $mg \cdot L^{-1}$ carbofuran (formulation not given) on selected biochemical responses in the air-breathing catfish *Clarias batrachus*. After 30-d exposure to 0.5 $mg \cdot L^{-1}$ carbofuran, calcium levels in blood serum exhibited a significant decrease, the activity of brain acetylcholinesterase was significantly inhibited, blood serum ammonia levels were significantly increased (corresponding to a lower rate of excretion), magnesium and sodium-potassium ATPase activity in gill and intestinal tissue were significantly inhibited, and the activities of serum acid phosphatase and alkaline phosphatase were significantly increased in the same tissues. Significant increases in the serum enzymes glutamate oxalacetate transaminase and glutamate pyruvate transaminase, and significant increases in the activity of glucose-6-phosphatase corresponding to decreases in liver glycogen and increased serum glucose were also observed. Despite these biochemical alterations (from 30-d exposure to 0.5 $mg \cdot L^{-1}$ carbofuran), only a slight reduction in growth rate was demonstrated after exposure to the same concentration for 60 d. Following exposure to 0.5 $mg \cdot L^{-1}$ carbofuran for 30 d, only traces of the insecticide were found in the liver. However, 3-hydroxycarbofuran was detected in measurable quantities although the concentrations were not reported.

The chronic exposure (120 d) to a presumably safe concentration of carbofuran (5 $mg \cdot L^{-1}$) was found to decrease the ovary weights in treated fish (*Channa punctatus*). Initial decreases were observed after 30 d and continued to decrease to a maximum at 120 d. Associated with the decrease in ovary weight were low levels of recrudescence (ovulation) and a low occurrence of vitellogenic and yolky oocytes in the ovaries. The observed treatment effects were thought to have been the result of carbofuran interference with steroid hormone production, particularly the gonadotrophins (Mani and Saxena, 1985).

In one of the more comprehensive chronic investigations with fish, Hansen and Parrish (1977) used the North American sheepshead minnow in long-term, life-cycle exposures. Based upon survival of parental fish, hatching success of eggs, and fry mortality and development, a maximum acceptable toxicant concentration of between 15 and 23 $\mu g \cdot L^{-1}$ was developed.

Invertebrates

In a study of the effects of pesticides on crawfish (*Procambarus clarkii*) production in rice fields, Graves et al. (1977) concluded that a broadcast application of carbofuran granules (93% ai) at 0.56 $kg \cdot ha^{-1}$ did not influence crawfish size, sex-ratio, or weight yield from treated plots.

Grant et al. (1983) examined the effectiveness of carbofuran (100% ai) to reduce the grazing of ostracods (*Cyprinotus carolinensis* and *Heterocypris luzonensis*) on blue-green algae. Grazing by both ostracod species was reduced by a concentration of 0.1 $mg \cdot L^{-1}$. Cessation of grazing in *C. carolinensis* occurred at 1.0 $mg \cdot L^{-1}$ and in *H. luzonensis* at 25 $mg \cdot L^{-1}$.

The freshwater clams *Glebula rotundata* and *Rangia cuneato* are apparently very tolerant of carbofuran. However, sublethal symptoms of toxicity (i.e., shell gaping, foot extension, and incoordination) were evident when carbofuran exposures were in the 75-$mg \cdot L^{-1}$ range (Zakour, 1980).

Plants

The toxic effect of carbofuran (as Furadan containing 3% ai) on the blue-green alga *Nostoc muscorum* was investigated by Kar and Singh (1978). The alga was found to tolerate 1000 $mg \cdot L^{-1}$ of the Furadan formulation in both nitrogen-containing and nitrogen-free media for 10-d periods. Higher concentrations, however, did not support growth in either algal growth medium. At a concentration of 25 $mg \cdot L^{-1}$, a slight growth stimulation was noted. Specific growth rate constants gradually decreased with increasing Furadan concentrations from 50 to 1000 $mg \cdot L^{-1}$. Ten-day toxicity tests demonstrated that a concentration of 1200 $mg \cdot L^{-1}$ was algastatic, whereas higher concentrations were algicidal.

Enhancement of heterocyst frequency (as a consequence of increased nitrogen fixation) occurred at the lower Furadan concentrations used (25 $mg \cdot L^{-1}$ or 0.75 $mg \cdot L^{-1}$ carbofuran). Suppression of heterocyst frequency by 50 to 1000 $mg \cdot L^{-1}$ during 10-d incubation periods was interpreted by the authors as the use of carbofuran as a nitrogen source. In addition, the authors argued for a possible metabolism or biodegradation of carbofuran by

the algae. The cultures of algae used, however, were not described as axenic, and therefore it was hard to give this argument any merit (Kar and Singh, 1978).

In a subsequent study, Kar and Singh (1979), also using *Nostoc muscorum*, reported growth enhancement by 25 $mg \cdot L^{-1}$ in the pH range of 5–10. This enhanced growth was produced under a light intensity of 3000 lx, but did not occur at 1500 lx, where inhibition was observed with 25 $mg \cdot L^{-1}$. Compared to control algal cultures grown at the same pH, the higher concentrations of carbofuran (i.e., 100 and 500 $mg \cdot L^{-1}$) were more toxic at acidic pHs (5.6) than at alkaline pHs (7.5–10). The authors did not discuss the relative stability of carbofuran at acid and alkaline pHs. The algal population densities used also had an effect on the toxic effect of carbofuran. Increased population densities tended to decrease toxic effects at all levels.

Guideline

While it is evident from Appendix B that a data base of acute toxicity exists for North American freshwater species, the previous discussion concerning sublethal reactions and chronic toxicity demonstrates that the majority of the work has been conducted on fish species native to the Indian subcontinent. There is very little known concerning sublethal reactions and chronic toxicity to sensitive North American aquatic species.

It is apparent that carbofuran is highly toxic to aquatic organisms, with acute effects on fish and invertebrates occurring at water concentrations as low as 0.08 $mg \cdot L^{-1}$ and 0.035 $mg \cdot L^{-1}$, respectively. The upper limit of a maximum acceptable toxicant concentration derived from chronic toxicity tests on a North American fish species is 0.023 $mg \cdot L^{-1}$.

Specific Canadian objectives or guidelines for carbofuran in water for the protection of aquatic life do not exist. In accordance with the CCREM-prescribed guideline development procedures, a Canadian water quality guideline for aquatic life is derived by using the lowest no-effect estimate (MATC, NOEL, or equivalent) with a safety factor of one order of magnitude. In the absence of such reliable data, the alternate approach is to apply the appropriate application factor (AF) to the lowest LC_{50} value available for a Canadian species.

In the case of the sheepshead minnow, MATC range is not used as the basis for the guideline because of its typical estuarine distribution and nonendemic status in Canada. The lowest acute toxicity value derived in an approved manner is the 48-h LC_{50} of 0.035 $mg \cdot L^{-1}$ for the cladoceran *Daphnia pulex*. Using this value and the application factor of 0.05 (nonpersistent pesticides) generates a guideline for the protection of aquatic life of 1.7 $\mu g \cdot L^{-1}$.

Aquatic animals are among the organisms most sensitive to carbofuran toxicity and it is therefore recommended that the water quality guideline for the protection of aquatic organisms be established at 1.7 $\mu g \cdot L^{-1}$. This is the upper limit of the range of values derived by application factors in current use for pesticides in general. It is below all known values initiating sublethal physiological and biochemical responses in fish. A value of 1.7 $\mu g \cdot L^{-1}$ also takes into account the increased sensitivity of some fish to carbofuran relative to the maximum acceptable toxicant concentration established by the sheepshead minnow. In addition, the guideline is above established detection limits (0.1 $\mu g \cdot L^{-1}$) for carbofuran in water (Environment Canada, 1979). Accurate and precise quantification should not, therefore, be a problem.

Agricultural Uses

Livestock Watering

Acute Toxicity to Livestock

Most studies indicate that bird and mammal poisonings are a result of direction ingestion of carbofuran granules or contaminated food rather than a result of ingestion of contaminated water.

Acute oral toxicities for birds and mammals are given in Tables 4 and 5. The 14-d oral toxicities for birds range from 238 to 38 900 $\mu g \cdot kg^{-1}$. Oral toxicities (as a single lethal dose) in mammals range from 2000 to 34 500 $\mu g \cdot kg^{-1}$. A review of carbofuran toxicity to birds and mammals was provided by Eisler (1985). Acutely toxic symptoms of carbofuran poisoning in birds include loss of muscular coordination, wings crossed high over the back, head nodding, vocalization, salivation, tears, diarrhea, immobility with wings spread, laboured breathing, pupil constriction, arching of back, and/or arching of the neck over the back.

Table 4. Acute Oral Toxicities of Carbofuran to Birds and Mammals

Taxonomic group and species tested	14-d LD_{50} ($\mu g \cdot kg^{-1}$)*	Reference
Bird		
Fulvous whistling-duck (*Dendrocygna bicolor*)	238	Tucker and Crabtree, 1970
Mallard (*Anas platyrhynchos*)		
Age 36 h	280–480	Hudson et al., 1972
Age 7 d	530–740	Hudson et al., 1972
Age 30 d	410–640	Hudson et al., 1972
Age 3–4 months	320–500	Tucker and Crabtree ,1970
Age 6 months	330–520	Hudson et al., 1972
Red-winged blackbird (*Agelaius phoeniceus*)	422	Schafer et al., 1973
Quelea (*Quelea quelea*)	422–562	Schafer et al., 1973
House finch (*Carpodacus mexicanus*)	750	Schafer et al., 1973
Japanese quail (*Coturnix japonica*)	1300–2100	Sherman and Ross, 1969
House sparrow (*Passer domesticus*)	1330	Schafer et al., 1973
Common grackle (*Quiscalus quiscula*)	1330–3160	Schafer et al., 1973
Rock dove (*Columba livia*)	1330	Schafer et al., 1973
Brown-headed cowbird (*Molothrus ater*)	1330	Schafer et al., 1973
Ring-necked pheasant (*Phasianus colchicus*)	2380–7220	Tucker and Crabtree, 1970
Bobwhite quail (*Colinus virginianus*)	3640–6990	Tucker and Crabtree, 1970
European starling (*Sturnus vulgaris*)	5620	Schafer et al., 1973
Mammals		
Mouse	2000	NRCC, 1979
Cat	2500–3500	NRCC, 1979
Old-field mouse	4000	Wolfe and Esher, 1980
Sheep	8000	Palmer and Schlinke, 1973
Guinea pig	9200	NRCC, 1979

Source: Eisler, 1985.

*Concentrations shown are in micrograms carbofuran administered per kilogram body weight ($\mu g \cdot kg^{-1}$) in a single dose fatal to 50% within 14 d.

Table 5. Acute Toxicity of Dietary Carbofuran to Birds and Mammals

Organism	Concentration*	Exposure Interval (d)		Mortality (%)	Reference
		Exposure	Post-exposure		
Birds					
Mallard					
(*Anas platyrhynchos*)	190	5	3	50	Hill et al., 1975
	242	5	3	50	Kononen et al., 1987
	23 (as water)	5	3	50	Kononen et al., 1987
Ring-necked pheasant					
(*Phasianus colchicus*)	573	5	3	50	Hill et al., 1975
Japanese quail					
(*Coturnix japonica*)					
Age 36 h	140–471	5	3	50	Hill and Camardese, 1983
Age 7 d	436–1103	5	3	50	Hill and Camardese, 1983
Age 10–14 d	438	5	3	50	Hill et al., 1975
Age 14 d	586–1004	5	3	50	Hill and Camardese, 1983
Age 21 d	779–1459	5	3	50	Hill and Camardese, 1983
Bobwhite quail					
(*Colinus virginianus*)	917	5	3	50	Kononen et al., 1987
	>100	5	3	50	Kononen et al., 1987
	(as water)				
Mammals					
Old-field mouse					
(*Peromyscus polionotus*)	500	4		100	Wolfe and Esher, 1980
	250	4		20	
	100	240		38	

Source: Eisler, 1985, with additional data from Kononen et al., 1987.

*Concentration of carbofuran in diet (i.e., solid food) in $mg \cdot kg^{-1}$ fresh weight unless otherwise stated.

While carbofuran may cause acutely toxic reactions in birds by way of the respiratory and dermal routes, oral exposure appears to be the most important route. In particular, the consumption of contaminated vegetables, poisoned vertebrates and invertebrates, and the granular formulations of carbofuran constitute the most numerous examples of bird poisoning (Eisler, 1985).

Flickinger et al. (1986) reported the poisoning of birds (mainly passerines) in an east Texas rice field apparently treated with Furadan 4F during planting. Cursory examination revealed a total of 106 dead birds (comprising 11 species). Subsequent analysis of brain cholinesterase activity from carcasses revealed a greater than 50% reduction in activity in 31% of the birds. This is diagnostic of death from anticholinesterase agent (such as carbofuran) poisoning. Carbofuran residues in the gastrointestinal tracts of the dead birds averaged 3.4 $mg \cdot kg^{-1}$ and ranged from 0 to 10 $mg \cdot kg^{-1}$.

As well, the deaths of songbirds (order Passeriformes) have been attributed to the field application of carbofuran granules to corn fields and orchards. The dead birds had high levels of carbofuran in their livers and gastrointestinal tracts apparently as the result of intensive feeding in the treated fields. Laboratory studies have demonstrated that ingestion of a single carbofuran granule of Furadan 10G (i.e., 10% ai) can be lethal to house sparrows (*Passer domesticus*) and red-winged blackbirds (*Agelaius phoeniceus*). Balcomb et al. (1984) reported the ingestion of Furadan 10G at 2.3 $mg \cdot ai \cdot kg^{-1}$ caused 50% mortality in *A. phoeniceus*. Carbofuran in dietary concentrations of 140–1459 $mg \cdot kg^{-1}$ produced 50% mortality in Japanese quail (*Coturnix japonica*) when consumed for 5 d. Reports also exist of raptors poisoned with carbofuran as the result of feeding on the carcasses of carbofuran-poisoned small mammals and birds (Eisler, 1985).

In contrast to the above field and laboratory studies, the ingestion of carbofuran-contaminated water has received little attention. It is known that ingestion of water containing 1 $mg \cdot L^{-1}$ carbofuran by ducks resulted in toxic symptoms after 7 d. A 2-$mg \cdot L^{-1}$ dose in the drinking water was lethal in 7 d (Eisler, 1985).

Mammals are generally less sensitive to the acute toxicity of orally administered carbofuran than birds. As with birds, however, much of the mammalian toxicity data deals with the ingestion of solid material contaminated with carbofuran as opposed to the ingestion of water containing carbofuran.

Osheim et al. (1985) reported the death of seven crossbred Brahman cattle that accidentally ingested carbofuran-treated rice (0.946 L of 40.64% carbofuran to 38 L of rice). Three cows died within 20 min of ingesting the rice. Ten cows exhibited varying clinical symptoms of carbofuran poisoning including salivation, respiratory distress, diarrhea, staggering gait, and recumbency. Of these ten cows, four died despite an intravenously injected antidote (atropine sulphate). The remaining six cows recovered and were free of carbofuran poisoning symptoms 6 h after exposure.

Levels of carbofuran in the rumen contents of the dead cows were found to be between 2 and 51 $mg \cdot kg^{-1}$ rumen contents. However, 1 week passed between the death of the cattle and rumen contents sampling, and reduction in carbofuran content was expected. The lethal dose of carbofuran for adult cattle is 18 $mg \cdot kg^{-1}$ body weight (Osweiler et al., 1985).

Subacute and Chronic Toxicity to Livestock

While acutely toxic reactions to carbofuran are the most dramatic responses observed in the field, much less is known concerning subacute and chronic exposures. Laboratory studies have established that single oral doses of 2 $mg \cdot kg^{-1}$ body weight administered to bobwhite quails (*Colinus virginianus*) did not affect brain cholinesterase levels at 48 h. Growth, metabolic efficiency, and energy metabolism were also unaffected after 8 d (Solomon and Robel, 1980).

The only observed chronic response exhibited by bobwhite quails fed carbofuran at 131 $mg \cdot kg^{-1}$ body weight for 14 d was reduced activity. Once placed on a carbofuran-free diet, this response disappeared (Robel, et al., 1983).

The avoidance of carbofuran-contaminated food and water by 10-d–old mallard ducks (*Anas platyrhynchos*) and bobwhite quails was quantified in a 5-d exposure, 3-d post-exposure study by Kononen et al. (1987). The highest concentration of carbofuran that allowed equal consumption of carbofuran-contaminated and uncontaminated food by a population having free access to both was determined to be a food avoidance concentration 50 or FAC_{50}. Discrimination between contaminated and uncontaminated food theoretically occurs above this level. The same rationale was employed to determine a water avoidance concentration 50 or WAC_{50} for the two species. Using a range of sublethal to lethal dietary concentrations of carbofuran in food and water, the FAC_{50}s and WAC_{50}s for mallard ducks were calculated to be 10 $\mu g \cdot g^{-1}$ and 3 $mg \cdot L^{-1}$, respectively. The FAC_{50} and WAC_{50} for bobwhite quail were 159 $\mu g \cdot g^{-1}$ and greater than 50 $mg \cdot L^{-1}$, respectively.

The difference between the values for the FAC_{50} and the WAC_{50} in the mallard was related to the capability of 1-d–old mallards to avoid food, but not water during the 5-d exposure period, and the higher water use (for consumption and preening) by mallards compared with food consumption. The result was a greater daily intake of water (which had a lower carbofuran content) than food on a weight/weight basis.

The same reasoning does not apply as well to the bobwhite quail. This species is much less sensitive to carbofuran than mallards, and sufficiently high concentrations of carbofuran in the drinking water could not be achieved for an adequate estimation of a WAC_{50} (Kononen et al., 1987).

In large mammals, such as cattle and sheep, the subacute toxic reactions include increased salivation, muscle tremors, prostration, laboured breathing, and loss of appetite.

These symptoms were observed in 1- to 2-week–old calves, cattle yearlings, and sheep given single oral doses of carbofuran at 0.25–5.0 $mg \cdot kg^{-1}$ body weight, 1.0–5.0 $mg \cdot kg^{-1}$ body weight, and 2.5–5.0 $mg \cdot kg^{-1}$ body weight, respectively (Palmer and Schlinke, 1973).

Several investigations have focused on possible endocrine dysfunction as a sublethal

response to carbofuran ingestion. These studies have used steroid metabolism as monitors of potentially detrimental sublethal responses. *In vitro* studies of testosterone metabolism by rat and mouse liver and prostate tissues have demonstrated only a slight stimulation of microsomal hydroxylation reactions by 22.1 $mg \cdot L^{-1}$ carbofuran (technical grade). The anticipated detrimental response was to have been a decrease in hydroxylation reactions, as is produced by other types of insecticides (Schein, et al., 1976; Donovan et al., 1978).

Another endocrine dysfunction study used the ingestion (12.3 mg per week) of carbofuran (technical grade) by mice for a period of 90 d. The consumption of carbofuran at this concentration (30 $\mu g \cdot g^{-1}$ in food) did not affect food consumption or body weight gain in the experimental animals. At the end of 90 d, carbofuran was found to have significantly affected prostate weight, prostate protein content, and prostate RNA and DNA content. These alterations of endocrine function were, however, termed physiologically insignificant (Shain et al., 1977).

Immune system responses to the ingestion of carbofuran have also been examined in the offspring of female mice receiving carbofuran (technical grade) doses of 0.01 and 0.50 $mg \cdot kg^{-1} \cdot d^{-1}$ for the period of gestation (approximately 22 d). Treatment effects were most apparent (as decreased or increased serum immunoglobulins) in the offspring at 3 months of age, but systematic dose-effect relationships were not apparent. Females exposed to 0.01 $mg \cdot kg^{-1} \cdot d^{-1}$ produced female offspring with increased plasma corticosterone and decreased total hepatic metabolism of corticosterone. Females exposed to 0.50 $mg \cdot kg^{-1} \cdot d^{-1}$, however, produced offspring with apparently normal endocrine function as defined by hepatic corticosterone metabolism (Cranmer et al., 1978, 1979).

Other laboratory studies have also demonstrated carbofuran's subacute toxic response in mammals. Gupta and Bagchi (1982) demonstrated the effects of Furadan (percent active ingredient unknown) on mice by observing various behavioural responses. Furadan (in 0.9% NaCl) was administered by intraperitoneal injection at doses of 0.5, 0.75, and 1.0 $mg \cdot kg^{-1}$ body weight. Qualitative observations were made on the ability of the individual mice to turn over after being placed on their backs, the induction of a corneal reflex, the pinna reflex, and the grip strength of the tail and forepaws. Under the influence of the concentrations used, Furadan was observed to have a slight depressant action on the central nervous system.

Due to the reported antiacetylcholinesterase activity of carbofuran, a common method of studying subacute exposure has been the determination of this enzyme activity and other associated neurotransmitters. Changes in the concentrations of brain neurotransmitters (acetylcholine, catecholamines, and amino acids) after the intraperitoneal injections of Furadan (percent active ingredient not given) at a concentration of 0.25 $mg \cdot kg^{-1}$ were described by Gupta et al. (1984). Significant decreases in acetylcholinesterase activity were accompanied by significant increases in acetylcholine, gamma-amino-butyric acid, epinephrine, norepinephrine, dopamine, and 5-hydroxytryptamine concentrations. Apparently, the excitatory action of increased levels of acetylcholine is antagonistic to the action of gamma-amino-butyric acid, with a resulting depressant effect on the central nervous system. Epinephrine and norepinephrine are also known to cause central nervous system depression.

Uptake, Metabolism, and Depuration

Various laboratory studies (Knaak et al., 1970) have demonstrated that carbofuran is readily absorbed from the gastrointestinal tract of mammals. Experiments designed to study the rat gastrointestinal uptake of radiolabelled carbofuran from an emulphor-ethanol-water carrier demonstrated that 51.2% of the carbofuran was adsorbed in 15 min; 67.4% was absorbed in 60 min. As would be expected, most of the absorption of carbofuran appears to take place in the small intestine (Ahdaya et al., 1981; Ahdaya and Guthrie, 1982).

Other examinations of the uptake of carbofuran and the metabolites 3-hydrocarbofuran (3-OH-CF) and 3-hydroxycarbofuran glucoside (3-OH-CF-glucoside) demonstrated rapid absorption of carbofuran and 3-OH-CF in all segments of the rat gastrointestinal tract. By contrast, 3-OH-CF-glucoside was not readily absorbed until reaching the lower portion of the small intestine and cecum where the glucosidic linkage is cleaved and the resulting 3-OH-CF is absorbed (Marshall and Dorough, 1979).

Limited data on the dermal absorption of carbofuran by mice indicate that 33% of the applied carbofuran can be absorbed through the skin in 5 min. Seventy-six percent can be absorbed in 60 min

and 95% can be absorbed in 480 min (Shah et al., 1981).

Once in the bloodstream, the liver becomes the primary organ for carbofuran metabolism as the result of reactions involving oxidation, reduction, hydrolysis, and conjugation. Prior to metabolism, carbofuran may exert an inhibitory effect on acetylcholinesterase activity. Laboratory studies using sublethal oral doses (i.e., 50 μg carbofuran·kg^{-1} body weight) demonstrated a short enzyme recovery period due primarily to the kinetics of the reversible acetylcholinesterase-carbofuran complex. The rate of complex formation is directly correlated with carbofuran toxicity. Recovery from inhibition simply requires the dissociation of the methylcarbamyl moiety from the enzyme (Ferguson et al., 1984). Oral exposure produces rapid acetylcholinesterase recovery and less acetylcholinesterase depression compared to other routes of exposure (e.g., dermal, inhalation, intravenous) due to the complete biotransformation of the insecticide.

An important plasma and urine metabolite of carbofuran is 3-hydroxycarbofuran. It is rapidly formed and distributed among the various tissues after carbofuran's first pass through the enterohepatic tissue. Its half-life in the mammalian system ($t_{1/2}$ = 64 min), however, is approximately twice as long as the parent carbofuran molecule ($t_{1/2}$ = 29 min). Although, the 3-hydroxycarbofuran metabolite has acetylcholinesterase activity similar to the parent compound, the rapid metabolic conjugation of this metabolite may be the more important aspect of its *in vivo* disposition (Ferguson et al. 1984). The hydroxylation of carbofuran to 3-hydroxycarbofuran and the hydrolysis of the carbamate ester linkage are also the predominant pathways of carbofuran in laying hens. A single oral dose of 2.7 mg·kg^{-1} was subject to rapid hydrolytic degradation. After approximately 6 h, 54% of the dose had been hydrolyzed. Twenty-six percent of the dose, approximately half of which were metabolic products, was eliminated in the feces in the same time frame. The metabolism of carbofuran reached near stabilization after 24 h. By this time, 72% of the dose had been hydrolyzed; 66% was eliminated in the feces and 12% remained in the body as carbofuran metabolites (Hicks et al., 1970).

The toxicity of other carbofuran metabolites is significantly less than the parent compound (NRCC, 1979; Ferguson et al., 1984). Metabolism of 3-hydroxycarbofuran and 3-hydroxycarbofuran glucoside, after their formation in the liver from the parent carbofuran, yield the same breakdown products in the bile. Metabolism of the glucoside conjugate, however, is much slower than the unconjugated 3-hydroxycarbofuran (Dorough, 1983).

Depuration studies with rats indicated rapid (i.e., approximately 75%) excretion of carbofuran metabolites within the first 24 h after oral administration (Dorough, 1968). Elimination studies with radiolabelled carbofuran showed that 24% of the oral dose of carbofuran was found in the urine after 60 min. Six percent of the radioactive dose appeared in the air respired from the lungs within the same time frame (Ahdaya et al., 1981).

Mutagenicity, Teratogenicity, and Carcinogenicity

Short-term mutagenic assays of carbofuran have produced few positive results. Dogs, rats, and rabbits have been used to assess the reproductive and teratogenic effects of carbofuran. Teratogenic effects were not observed, but testicular degeneration was noted. No-effect levels for testicular degeneration were 1.0 and 2.0 mg·kg^{-1}·d^{-1} for rats and rabbits, respectively (Life Systems, Inc., 1985).

Many organic compounds containing the N-nitroso group (N=O) are known mutagens, and concern has been raised by some investigators concerning the creation of N-nitrosocarbofuran or N-nitrosocarbofuran metabolites. The creation of nitroso derivatives attains its greatest potential in the acid condition of mammalian stomachs. Investigations of *in vivo* nitrosocarbofuran formation in the stomachs of rats and guinea pigs appeared to demonstrate only low levels of the nitroso derivatives (i.e., 0.65% and 0.03% in guinea pig and rat stomachs, respectively). Higher yields were obtained with *in vitro* mixtures of carbofuran, sodium nitrate, and gastric contents (Rickard and Dorough, 1984). The nitroso derivatives of carbofuran or its metabolites have been shown to be mutagenic when tested with the Ames Test (Nelson et al., 1981). Doses of N-nitrosocarbofuran (by gastric intubation) of 16.5 mg·kg^{-1} given once a week for 23 weeks were found to cause increased stomach tumors in rats. Lower dosage gave less conclusive results (Lijinsky and Schmahl, 1978). There is no firm evidence, however, for the biological or environmental formation of N-nitrosocarbofuran or N-nitrosocarbofuran metabolites, which could pose either a mutagenic or carcinogenic threat to animal life (Life Systems, Inc., 1985).

Guideline

Information specifically related to carbofuran in livestock water supplies could not be found. Ontario has a 0.1-mg·L^{-1} water quality objective for the general category of carbamate and organophosphorus insecticides in livestock watering supplies (Ontario Ministry of the Environment, 1984).

It is significant that most studies indicate that mammal and bird poisonings are a result of direct ingestion of carbofuran granules or contaminated food rather than the result of contaminated water ingestion. The guideline for livestock watering should take into account the maximum acceptable daily intake for humans (90 μg·L^{-1}) derived from a 2-year laboratory study of carbofuran intake by rats with a no-observed-effect level of 0.01 mg·kg^{-1}·d^{-1} (Health and Welfare Canada, 1987). Although the 90-μg·L^{-1} value would conceivably be safe for livestock of greater body weight than humans, small animals might be at risk. Available data suggest that sensitive bird species might not be protected at a level of 90 μg·L^{-1}. An oral LD_{50} of 0.42 mg·kg^{-1} for red-winged blackbirds indicates a considerably higher degree of sensitivity to carbofuran than mammals (Schafer et al., 1973). This study implies that 27.3 μg of carbofuran ingested orally would be lethal to half a population over 96 h. To introduce a margin of safety, the value of 90 μg·L^{-1} is reduced by half to 45 μg·L^{-1} for a recommended guideline.

Irrigation

Guideline

Specific information regarding carbofuran toxicity to terrestrial or aquatic vascular plants could not be found in the literature. As well, there are no data to suggest that carbofuran residues in irrigation water that result from registered uses are harmful to crops. Therefore, no guideline is recommended.

Recreational Water Quality and Aesthetics

Guideline

There is no recommended guideline for carbofuran in recreational waters.

Industrial Water Supplies

Guideline

There is no recommended guideline for carbofuran in industrial water supplies.

SUMMARY

Following an extensive evaluation of the published literature on the pesticide carbofuran, Canadian water quality guidelines were derived (Table 6). The background information on carbofuran in terms of uses and production, occurrence in the aquatic environment, and persistence and degradation was reviewed. The rationale employed for the development of the recommended guidelines was summarized.

Table 6. Recommended Water Quality Guidelines for Carbofuran

Uses	Guidelines
Raw water for drinking water supply	90 μg·L^{-1}*
Freshwater aquatic life	1.7 μg·L^{-1}
Agricultural uses	
Livestock watering	45 μg·L^{-1}
Irrigation	No recommended guideline
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

*Existing drinking water guideline (Health and Welfare Canada, 1987).

ACKNOWLEDGMENTS

The authors gratefully acknowledge the assistance and comments of the members of the Canadian Council of Resource and Environment Ministers (CCREM) Task Force on Water Quality Guidelines and the scientific reviewers from Environment Canada (R.C. Pierce and M.C. Taylor of the Water Quality Branch, J. Maguire and W. Strachan of the National Water Research Institute, P. Mineau and K. Lloyd of the Canadian Wildlife Service, H. Lerer and M. Constable of Environmental Protection), V. Zitko of Fisheries and Oceans Canada, and G. Wood of Health and Welfare Canada.

REFERENCES

Ahdaya, S., and F.E. Guthrie. 1982. Stomach absorption of intubated insecticides in fasted mice. Toxicology. 22: 311-317.

Ahdaya, S.M., R.J. Monroe, and F.E. Guthrie. 1981. Absorption and distribution of intubated insecticides in fasted mice. Pestic. Biochem. Physiol., 16: 38-46.

Albanis, T.A., P.J. Pomonis, and A. Th. Sdoukos. 1986. Organo-phosphorous and carbamate pesticide residues in the aquatic system of Ioannian basin and Kalamas River (Greece). Chemosphere, 15: 1023-1034.

Bailey, H.S. 1984. Survey of toxic constituents in the Atlantic region's aquatic environment—1982. IWD-AR-WQB-84-55. Water Quality Branch, Atlantic Region, Inland Waters Directorate, Environment Canada, Moncton.

Bailey, H.S. 1985. 1983 Toxic chemical survey: A survey of nine impoundments in the Saint John River basin. IWD-AR-WQB-85-88. Water Quality Branch, Atlantic Region, Inland Waters Directorate, Environment Canada, Moncton

Bailey, H.S., and G.D. Howell. 1983. Survey of toxic organic constituents in the Atlantic region's aquatic environment (1979/81). IWD-AR-WQB-83-48. Water Quality Branch, Atlantic Region, Inland Waters Directorate, Environment Canada, Moncton.

Bakthavathsalam, R., and Y.S. Reddy. 1981. Lipid kinetics in relation to the toxicity of three pesticides in the climbing perch, *Anabas testudineus* (Bloch). Proc. Indian Nat. Sci. Acad. Part B. 47(1): 670-676.

Bakthavathsalam, R., and Y.S. Reddy. 1983. Impact of Furadan on the succinate dehydrogenase activity of an air-breathing fish, *Anabas testudineus* (Bloch). Proc. Indian Nat. Sci. Acad. Part B. 49: 9-14.

Bakthavathsalam, R., and Y.S. Reddy. 1985. Toxic effects of Disyston and Furadan on the bimodal pattern of oxygen consumption in the climbing perch, *Anabas testudineus* (Bloch). Water Res., 19: 1195-1198.

Bakthavathsalam, R., R. Ramalingam, and A. Ramaswamy. 1984. Histopathology of liver, kidney and intestine of the fish *Anabas testudineus* exposed to Furadan. Environ. Ecol., 2(4): 243-247.

Balcomb, R., R. Stevens, and C. Bowen, II. 1984. Toxicity of 16 granular insecticides to wild-caught songbirds. Bull. Environ. Contam. Toxicol., 33: 302-307.

Belluck, D., and A. Felsot. 1981. Bioconcentration of pesticides by egg masses of the caddisfly *Triaenodes tardus* Milne. Bull. Environ. Contam., Toxicol., 26: 299-306.

Bhattacharya, S. 1985a. Mechanism of toxic action of pesticides on enzymes and its reversal in fishes. Geobios, 12: 241-244.

Bhattacharya, S. 1985b. Toxicity of carbofuran and phenthoate in *Channa punctatus* and *Anabas testudineus*. J. Environ. Biol., 6(2): 129-137.

Bowman, B.T., and W.W. Sans. 1983. Determination of octanol-water partitioning coefficients (K_{ow}) of 61 organophosphorous and carbamate insecticides and their relationship to respective water solubility (S) values. J. Environ. Sci. Health Part B. 18: 667-683.

Bush, P.B., D.G. Neary, J.W. Taylor, Jr., and W.L. Nutter. 1986. Effects of insecticide use in a pine seed orchard on pesticide levels in fish. Water Resour. Bull., 22(5): 817-827.

Caro, J.H., H.P. Freeman, D.E. Glotfelty, N.C. Turner, and W.M. Edwards. 1973. Dissipation of soil-incorporated carbofuran in the field. J. Agric. Food Chem., 21: 1010-1015.

Carter, F.L., and J.B. Graves. 1973. Measuring effects of insecticides on aquatic animals. L. Agric., 16: 14-15. (Cited in NRCC. 1979.)

Cohen, S.Z., C. Elden, and M.N. Lorber. 1986. Monitoring ground water for pesticides. In Evaluation of Pesticides in Ground Water, ed. W.Y. Garner, R.C. Honeycutt, and H.N. Nigg. Am. Chem. Soc. Symp. Ser. No. 315, pp. 179-196.

Cohen, S.Z., S.M. Creeger, R.F. Carsel, and C.G. Enfield. 1984. Potential pesticide contamination of groundwater from agricultural uses. In Treatment and Disposal of Pesticide Wastes, ed. R.F. Krueger and J.N. Seiber. Am. Chem. Soc. Symp. Ser. No. 259, pp. 297-325.

Cranmer, J.S., D.L. Avery, and J.B. Barnett. 1979. Persistent postnatal effects of the immune system following prenatal exposure to the anticholinesterase pesticides diazinon or carbofuran. Toxicol. Appl. Pharmacol., 48: A88 (abstract).

Cranmer, J.S., D.L. Avery, R.R. Grady, and J.I. Kitay. 1978. Postnatal endocrine dysfunction resulting from prenatal exposure to carbofuran, diazinon or chlordane. J. Environ. Pathol. Toxicol., 2: 357-369.

Dad, N.K., S.A. Qureshi, and V.K. Pandya. 1982. Acute toxicity of two insecticides to tubificid worms, *Tubifex tubifex* and *Limnodrilus hoffmeisteri*. Environ. Int., 7: 361-363.

Davey, R.B., M.V. Meisch, and F.L. Carter. 1976. Toxicity of five ricefield pesticides to the mosquito fish, *Gambusia affinis*, and green sunfish, *Lepomis cyanellus*, under laboratory and field conditions in Arkansas. Environ. Entomol., 5: 1053-1056.

Deuel, L.E., Jr., J.D. Price, F.T. Turner, and K.W. Brown. 1979. Persistence of carbofuran and its metabolites, 3-keto and 3-hydroxy carbofuran, under flooded rice culture. J. Environ. Qual., 8(1): 23-26.

Donovan, M.P., L.G. Schein, and J.A. Thomas. 1978. Effects of pesticides on metabolism of steroid hormone by rodent liver microsomes. J. Environ. Pathol. Toxicol., 2: 447-454.

Dorough, H.W. 1968. Metabolism of Furadan (NIA-10242) in rats and houseflies. J. Agric. Food Chem., 16: 319-325.

Dorough, H.W. 1983. Toxicological significance of pesticide conjugates. J. Toxicol. Clin. Toxicol., 19: 637-659.

Dudley, D.R., and J.R. Karr. 1980. Pesticides and PCB residues in the Black Creek Watershed, Allen County, Indiana—1977-78. Pestic. Monit. J., 13(4): 155-157.

Eco/Log Week. 1986. Carbofuran found in irrigation water. Vol. 14, No. 33, August 22, 1986. Don Mills, Ont.: Corpus Information Services.

Eisler, R. 1985. Carbofuran hazards to fish, wildlife and invertebrates: A synoptic review. Contaminant Hazard Review Report No. 3. Biological Report 85 (1.3). U.S. Department of the Interior, Fish and Wildlife Service, Laurel, Md.

Ekanem, S.B., J.W. Avault, Jr., J.B. Graves, and H. Morris. 1981. Acute toxicity of Propanil, Ordram and Furadan to crawfish (*Procambarus clarkii*) when chemicals were combined and used alone. J. World Maric. Soc., 12: 373-383.

Environment Canada. 1979. Analytical Methods Manual. Inland Waters Directorate, Water Quality Branch, Ottawa.

Erickson, D., G.M. Pulsky, W.E. Kortsch, and W.A. Charnetski. 1977. Carbofuran degradation in southern Alberta pond and lake water. Alberta Environment, Edmonton.

Ferguson, P.W., M.S. Dey, S.A. Jewell, and R.I. Krieger. 1984. Carbofuran metabolism and toxicity in the rat. Fundam. Appl. Toxicol., 4: 14-21.

Ferreira, G.A.L., and J.N. Seiber. 1981. Volatilization and exudation losses of three N-methylcarbamate insecticides applied systematically to rice. J. Agric. Food Chem., 29: 93-99.

Ferris, S.A. 1985. Insecticide residues in surface waters of agricultural areas in Saskatchewan. Water Pollution Control Branch, Saskatchewan Environment, Regina.

Flickinger, E.L., C.A. Mitchell, D.H. White, and E.J. Kolbe. 1986. Bird poisoning from misuse of the carbamate Furadan in a Texas rice field. Wildl. Soc. Bull., 14: 59-62.

Frank, R., H.E. Braun, M.V.-H. Holdrinet, G.J. Sirons, and B.D. Ripley. 1982. Agriculture and water quality in the Canadian Great Lakes basin: V. Pesticide use in 11 agricultural watersheds and presence in stream water, 1975-1977. J. Environ. Qual., 11(3): 497-505.

Gill, S.S. 1980. *In vitro* metabolism of carbofuran by liver microsomes of the padifield fish *Trichogaster pectoralis*. Bull. Environ. Contam. Toxicol., 25: 697-701.

Graham, J.R. 1977. Letter dated September 30, 1977, from J.R. Graham, FMC Corporation, Middleport, New York, 14015, to E.A.H. Smith, Environmental Secretariat, National Research Council of Canada, Ottawa.

Grant, I.F., E.A. Egan, and M. Alexander. 1983. Pesticides to control ostracods grazing on blue-green algae. Soil Biol. Biochem., 15(2): 193-197.

Graves, J.B., K.M. Hyde, F.L. Bonner, P.E. Schilling, and J.F. Fowler. 1977. Effects of mirex on crawfish production in rice fields. L. Agric., 20: 8-11.

Gupta, M., and G.K. Bagchi. 1982. Behavioural pharmacology of Furadan and Nuvacron in mice. Indian J. Hosp. Pharm., 19: 136-141.

Gupta, M., G.K. Bagchi, S.D. Gupta, D. Sasmal, T. Chatterjee, and S.N. Dey. 1984. Changes in acetylcholine, catecholamines and amino acid in mice brain following treatment with Nuvacron and Furadan. Toxicology, 30: 171-175.

Hansen, D.J., and P.R. Parrish. 1977. Suitability of sheepshead minnows (*Cyprinodon variegatus*) for life-cycle toxicity tests. In Aquatic Toxicology and Hazard Evaluation, ed. F.L. Mayer and J.L. Hamelink. First Symposium. ASTM STP 634. pp. 117-126.

Hartman, W.A., and D.B. Martin. 1985. Effects of four agriculture pesticides on *Daphnia pulex*, *Lemna minor*, and *Potamogeton pectinatus*. Bull. Environ. Contam. Toxicol., 35: 646-651.

Health and Welfare Canada. 1987. Guidelines for Canadian Drinking Water Quality 1987. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Advisory Committee on Environmental and Occupational Health.

Hejduk, J., and Z. Svobodova. 1980. Acute toxicity of carbamate-based pesticides for fish. Acta Vet. Brno, 49: 251-257.

Hicks, B.W., H.W. Dorough, and R.B. Davis. 1970. Fate of carbofuran in laying hens. J. Econ. Entomol., 63: 1108-1111.

Hill, E.F., and M.B. Camardese. 1983. Subacute toxicity testing with young birds: Response in relation to age and interest variability of LC-50 estimates. In Avian and Mammalian Wildlife Toxicology: 2nd Conference, ed. D.W. Lamb and E.E. Kenaga. ASTM Spec. Tech. Publ. 757. pp. 41-65. Philadelphia, Pa.: American Society for Testing and Materials.

Hill, E.F., R.G. Heath, W.J. Spann, and J.S. Williams. 1975. Lethal dietary toxicities of environmental pollutants to birds. U.S. Fish and Wildlife Serv. Spec. Sci. Rep. Wildlife 191. U.S. Department of the Interior, Washington, D.C.

Hiltibran, R.C. 1982. Effects of insecticides on the metal-activated hydrolysis of adenosine triphosphate by bluegill liver mitochondria. Arch. Environ. Contam. Toxicol., 11: 709-717.

Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1972. Effect of age on sensitivity: Acute oral toxicity of 14 pesticides to mallard ducks of several ages. Toxicol. Appl. Pharmacol., 22: 556-561.

Isensee, A.R., and N. Tayaputch. 1986. Distribution of carbofuran in a rice paddy-fish microecosystem. Bull. Environ. Contam. Toxicol., 36(5): 763-769.

Jash, N.B., and S. Bhattacharya. 1983. Delayed toxicity of carbofuran in freshwater teleosts *Channa punctatus* (Bloch) and *Anabas testudineus* (Bloch). Water Air Soil Pollut., 19: 209-213.

Johnson, B.T. 1986. Potential impact of selected agricultural chemical contaminants on a northern prairie wetland: A microcosm evaluation. Environ. Toxicol. Chem., 5: 473-485.

Johnson, W.W., and M.T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. Fish and Wildlife Service Resources Publication 137. U.S. Department of the Interior, Washington, D.C.

Kadoum, A.M., and D.E. Mock. 1978. Herbicide and insecticide residues in tailwater pits: Water and pit bottom soil from irrigated corn and sorghum fields. J. Agric. Food Chem., 29(1): 45-50.

Kar, S., and P.K. Singh. 1978. Toxicity of carbofuran to blue-green alga *Nostoc muscorum*. Bull. Environ. Contam. Toxicol., 20: 707-714.

Kar, S., and P.K. Singh. 1979. Effect of pH, light intensity, and population on the toxicity of the pesticide carbofuran to the blue-green alga *Nostoc muscorum*. Microbios, 21: 177-184.

Karnak, R.E., and W.J. Collins. 1974. The susceptibility to selected insecticides and acetylcholinesterase activity in a laboratory colony of midge larvae, *Chironomus tentans* (Diptera: Chironomidae). Bull. Environ. Contam. Toxicol., 12: 62-69.

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotoxicol. Environ. Saf., 4: 26-38.

Klaassen, H.E., and A.M. Kadoum. 1979. Distribution and retention of atrazine and carbofuran in farm pond ecosystems. Arch. Environ. Contam. Toxicol., 8: 345-353.

Knaak, J.B., D.M. Munger, J.F. McCarthy, and L.O. Satter. 1970. Metabolism of carbofuran alfalfa residues in the dairy cow. J. Agric. Food Chem., 18: 832-837.

Koeppe, M.K., and E.P. Lichtenstein. 1982. Effects of percolating water, captafol, and EPTC on the movement and metabolism of soil-applied [^{14}C] carbofuran in an agromicrocosm. J. Agric. Food Chem., 30: 116-121.

Konar, S.K., and T.K. Ghosh. 1983. Effects of some pesticides in mixture on fish, plankton and worm. Geobios, 10: 104-107.

Kononen, D.W., J.R. Hochstein, and R.K. Ringer. 1987. Avoidance behaviour of mallards and northern bobwhite exposed to carbofuran-contaminated food and water. Environ. Toxicol. Chem., 6: 41-50.

Krawchuk, B.P., and G.R.B. Webster. 1987. Movement of pesticides to ground water in an irrigated soil. Water Pollut. Res. J. Can., 22(1): 129-146.

Kulshrestha, S.K., and N. Arora. 1986. Effect of carbofuran, dimethoate and DDT on early egg development of *Cyprinus carpio*, Linn. Part I: Egg mortality and hatching. J. Environ. Biol., 7(2): 113-119.

Kulshrestha, S.K., N. Arora, and S. Sharma. 1986. Toxicity of four pesticides on the fingerlings of Indian major carps *Labeo rohita*, *Catla catla*, and *Cirrhinus mrigala*. Ecotoxicol. Environ. Saf., 12: 114-119.

Lemley, A.T., W.Z. Zhong, G.E. Janauer, and R. Rossi. 1984. Investigation of degradation rates of carbamate pesticides. Exploring a new detoxification method. In Treatment and Disposal of Pesticide Wastes., ed. R.F. Krueger and J.N. Seiber, Am. Chem. Soc. Symp. Ser. No. 259.

Life Systems, Inc. 1985. Drinking water criteria document for carbofuran. Final draft. Report to the Criteria and Standards Division, Office of Drinking Water, U.S. Environmental Protection Agency, Washington, D.C.

Lijinsky, W., and D. Schmahl. 1978. Carcinogenicity of N-nitroso derivatives of N-methylcarbamate insecticides in rats. Ecotoxicol. Environ. Saf., 2: 413-419.

Mani, K., and P.K. Saxena. 1985. Effect of safe concentrations of some pesticides on ovarian recrudescence in the freshwater murrel, *Channa punctatus* (Bl.): A quantitative study. Ecotoxicol. Environ. Saf., 9: 241-249.

Marshall, T.C., and H.W. Dorough. 1979. Comparative absorption and disposition of carbofuran, 3-hydroxycarbofuran and the glucoside of 3-hydroxycarbofuran. Toxicol. Lett. 48: A137 (abstract).

Mauck, B. 1972. Annual Report, 1972. Fish Pesticide Research Unit, Bureau of Sport Fishing and Wildlife, U.S. Department of the Interior, Lacrosse, Wis. (Cited in NRCC, 1979.)

Mayer, F.L., Jr., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Fish and Wildlife Service Resource Publication 160. U.S. Department of the Interior, Washington, D.C.

McCall, P.J., R.L. Swann, D.A. Laskowski, S.M. Unger, S.A. Vrona, and H.J. Dishburger. 1980. Estimation of chemical mobility in soil from liquid chromatographic retention times. Bull. Environ. Contam. Toxicol., 24: 190-195.

McGee, B. 1984. Survey of pesticide use in Ontario, 1983. Estimates of pesticides used on field crops, fruits, vegetables and in roadside weed control. Economics Information Report No. 84-05. Economics and Policy Coordination Branch, Ontario Ministry of Agriculture and Food, Toronto.

Metcalf, R.L., G.K. Sangha, and I.P. Kapoor. 1971. Model ecosystems for the evaluation of pesticide biodegradability and ecological magnification. Environ. Sci. Technol., 5: 709-713.

Metcalf, R.L., K.A. Reinbold, J.R. Sanborn, W.F. Childers, W.N. Bruce, and J. Coats. 1974. Comparative biochemistry, biodegradability and toxicity of DDT and carbofuran analogues. University of Illinois Water Resources Center Report No. 95. Urbana-Champaign, Ill.

Monenco Ltd. 1984. Atlantic provinces agricultural pesticide review. Report prepared for the Canadian Wildlife Service, Fredericton, N.B.

Mukhopadhyay, P.K., A.P. Mukherji, and P.V. Dehadrai. 1982. Certain biochemical responses in the air-breathing catfish *Clarias batrachus* exposed to sublethal carbofuran. Toxicology, 23: 337-345.

Neely, W.B., D.R. Branson, and G.E. Blau. 1974. Partition coefficient to measure bioconcentration potential of organic chemicals in fish. Environ. Sci. Technol., 8: 1113-1115.

Nelson, J., E.A. MacKinnon, H.F. Mower, and L. Wong. 1981. Mutagenicity of *N*-nitroso derivatives of carbofuran and its toxic metabolites. J. Toxicol. Environ. Health, 7: 519-531.

NRCC. 1979. Carbofuran: Criteria for interpreting the effects of its use on environmental quality. NRCC No. 16740. National Research Council of Canada, Ottawa.

Ontario Ministry of the Environment. 1984. Water management goals, policies, objectives and implementation procedures of the Ministry of the Environment. November 1978, revised May 1984, Toronto.

Osheim, D.L., P.F. Ross, L.D. Keck, and B.L. Tipton. 1985. Carbofuran toxicosis in cattle: Case history and analytical method. Vet. Hum. Toxicol., 27: 386-387.

Osweiler, G.D., T.L. Carson, W.B. Buck, and G.A. van Gelder. 1985. *Clinical and Diagnostic Veterinary Toxicology*. 3rd ed. Dubuque, Iowa: Kendall/Hunt Publishing.

Palmer, J.S., and J.C. Schlinke. 1973. Toxic effects of carbofuran in cattle and sheep. J. Am. Vet. Med. Assoc., 162: 561-563

Pawar, K.R., and M. Katdare. 1983a. Acute toxicity of Sumithion, BHC and Furadan to the freshwater prawn (*Macrobrachium rajdhari*). Geobios, 10: 136-137.

Pawar, K.R., and M. Katdare. 1983b. Toxicity of Sumithion, BHC and Furadan to the tadpoles of the frog *Microhyla ornata*. Indian J. Environ. Health, 25: 214-217.

Pawar, K.R., and M. Katdare. 1984. Effect of sublethal and lethal concentrations of fenitrothion, BHC and carbofuran on behaviour and oxygen consumption on the freshwater prawn *Macrobrachium kistnensis* (Tiwari). Arch. Hydrobiol., 99: 398-403.

Richards, R.P., J.W. Kramer, D.B. Baker, and K.A. Krieger. 1987. Pesticides in rainwater in the northeastern United States. Nature (London), 327: 129-131.

Rickard, R.W., and H.W. Dorough. 1984. *In vivo* formation of nitrosocarbamates in the stomach of rats and guinea pigs. J. Toxicol. Environ. Health, 14: 279-290.

Robel, R.J., R.W. Felthousen, and A.D. Dayton. 1983. Effects of dietary carbofuran on bobwhite activity patterns. Bull. Environ. Contam. Toxicol., 30: 105-109.

Sadhu, A.K., and P.K. Mukhopadhyay. 1985. Comparative effect of two pesticides malathion and carbofuran on testes of *Clarias batrachus* (Linn.). J. Environ. Biol., 6: 217-222.

Schafer, E.W., Jr., W.A. Bowles, Jr., and J. Hurlbert. 1983. The acute oral toxicity, repellency and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environ. Contam. Toxicol., 12: 355-382.

Schafer, E.W., Jr., R.B. Burton, N.F. Lockyer, and J.W DeGraziom. 1973. Comparative toxicity of seventeen pesticides to the quelea, house sparrow, and red-winged blackbird. Toxicol. Appl. Pharmacol., 26: 154-157.

Schein, L.G., J.A. Thomas, M.P. Donovan, and P.A. Klase. 1976. Effects of diazinon, parathion, and carbofuran on

the *in vitro* metabolism of ^{3}H-testosterone by rodent prostate glands and hepatic microsomal enzymes. Pharmacology. 18(2): 243 (abstract).

Selber. J.N., M.P. Catahan, and C.R. Barril. 1978. Loss of carbofuran from rice paddy water: Chemical and physical factors. J. Environ. Sci. Health Part B. 13(2): 131-148.

Sethunathan. N., T.K. Adhya, and K. Raghu. 1982. Microbial degradation of pesticides in tropical soils. In Biodegradation of Pesticides, ed. F. Matsumura and C.R.K. Murti, pp. 91-115. New York: Plenum Press.

Shah. P.V., R.J. Monroe, and F.E. Guthrie. 1981. Comparative rates of dermal penetration of insecticides in mice. Toxicol. Appl. Pharmacol., 59: 414-423.

Shain. S.A., J.C. Shaeffer, and R.W. Boesel. 1977. The effect of chronic ingestion of selected pesticides upon rat ventral prostate homeostasis. Toxicol. Appl. Pharmacol., 40: 115-130.

Sharom. M.S., J.R.W. Miles, C.R. Harris, and F.L. McEwen. 1980. Persistence of 12 insecticides in water. Water Res., 14: 1089-1093.

Sherman. M., and E. Ross. 1969. Acute and subacute toxicity to Japanese quail of the carbamate insecticides, carbofuran and SD 8530. Poult. Sci., 48: 2013-2018.

Siddaramappa. R., and J.N. Selber. 1979. Persistence of carbofuran in flooded rice soils and water. Prog. Water Technol., 11(6): 103-111.

Siddaramappa. R., and I. Watanabe. 1979. Evidence for vapor loss of ^{14}C-carbofuran from rice plants. Bull. Environ. Contam. Toxicol., 23: 544-551.

Siddaramappa. R., A.C. Tirol, J.N. Selber, E.A. Heinrichs, and I. Watanabe. 1978. The degradation of carbofuran in paddy water and flooded soil of untreated and retreated rice fields. J. Environ. Sci. Health Part B. 13(4): 369-380.

Solomon. K.E., and R.J. Robel. 1980. Effects of carbaryl and carbofuran on bobwhite energetics. J. Wildl. Manage., 44: 682-686.

Sur. R.K., and K.C. Ghose. 1978. Studies on *in vitro* and *in vivo* inhibition of fish brain cholinesterase by some insecticides. Indian J. Physiol. Allied Sci., 32(2): 72-78.

Thomson. W.T. 1979. *Agricultural Chemicals*. Book I. *Insecticides*. 1979-80 Revision. Fresno, Calif.: Thomson Publications.

Tucker. R.K., and D.G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. Fish and Wildlife Service Resource Publication 84. U.S. Department of the Interior, Washington, D.C.

Venkateswarlu. K., and N. Sethunathan. 1978. Degradation of carbofuran in rice soils as influenced by repeated applications and exposure to aerobic conditions following anaerobiosis. J. Agric. Food Chem., 26(5): 1148-1151.

Venkateswarlu. K., and N. Sethunathan. 1984. Fate of ^{14}C-carbofuran in a flooded acid sulphate saline soil. Curr. Sci., 53: 925-927.

Verma. S.R., S. Rani, S.K. Bansal, and R.C. Dalela. 1980. Effects of the pesticides thiotox, dichlorvos and carbofuran on the test fish *Mystus vittatus*. Water Air Soil Pollut., 13: 229-234.

Verma. S.R., S.R. Rani, and R.C. Dalela. 1981a. Isolated and combined effects of pesticides on serum transaminases in *Mystus vittatus* (African catfish). Toxicol. Lett., 8: 67-71.

Verma. S.R., S. Rani, and R.C. Dalela. 1981b. Pesticide-induced physiological alterations in certain tissues of a fish, *Mystus vittatus*. Toxicol. Lett., 9: 327-332.

Verma. S.R., S. Rani, S.K. Bansal, and R.C. Dalela. 1981. Evaluation of the comparative toxicity of thiotox, dichlorvos and carbofuran to two fresh water teleosts *Ophiocephalus punctatus* and *Mystus vittatus*. Acta Hydrochim. Hydrobiol., 9: 119-129.

Verma. S.R., S.K. Bansal, A.K. Gupta, N. Pal, A.K. Tyagi, M.C. Bhatnagar, V. Kumar, and R.C. Dalela. 1982. Bioassay trials with twenty-three pesticides to a fresh water teleost, *Saccobranchus fossilis*. Water Res., 16: 525-529.

Wauchope. R.D., and R.A. Leonard. 1980. Maximum pesticide concentrations in agricultural runoff: A semi-empirical prediction formula. J. Environ. Qual., 9(4): 665-672.

Wolfe. J.L., and R.J. Esher. 1980. Toxicity of carbofuran and lindane to the old-field mouse (*Peromyscus polionotus*) and the cotton mouse (*P. gossypinus*). Bull. Environ. Contam. Toxicol., 24: 894-902.

Worthing. C.R., and S.B. Walker (eds.). 1983. *The Pesticide Manual. A World Compendium*. 7th ed. The British Crop Protection Council, Croydon, U.K.

Yu. C-C., G.M. Booth, D.J. Hansen, and J.R. Larsen. 1974. Fate of carbofuran in a model ecosystem. J. Agric. Food Chem., 22(3): 431-434.

Zakour. H.R. 1980. Toxicity, uptake and metabolism of the N-methylcarbamate pesticide carbofuran by the freshwater bivalve mollusc *Glebula rotundata* (Lamarck). Ph.D. thesis, Rice University, Houston.

Appendix A
Summary of Carbofuran Residues Found in Aquatic Environments

Table A-1. Summary of Carbofuran Residues Found in Aquatic Environments

Location and conditions	Matrix	Samples with carbofuran/ samples collected	Concentration	Reference
26 tailwater pits in Haskell Co., KA, each receiving irrigation runoff water from one or more fields of corn. (Sampling conducted in 1973 and 1974.)*	Runoff pit soil	2/2	50 $\mu g \cdot kg^{-1}$ (mean), 75.9 $\mu g \cdot kg^{-1}$ (maximum)	Kadoum and Mock, 1978
	Runoff pit Water	10/12	35.2 $\mu g \cdot L^{-1}$ (mean), 88.9 $\mu g \cdot L^{-1}$ (maximum)	
5 tailwater pits in Haskell Co., KA, each receiving irrigation runoff water from corn and grain sorghum fields. (Sampling conducted from May 1975 to April 1977.)	Runoff pit soil	2/2	30.6 $\mu g \cdot kg^{-1}$ (mean), 42.4 $\mu g \cdot kg^{-1}$ (maximum)	
	Runoff pit Water	2/3	24.1 $\mu g \cdot L^{-1}$ (mean), 51.7 $\mu g \cdot L^{-1}$ (maximum)	
11 agricultural watersheds in southern Ontario. Carbofuran used in two watersheds for a total of 1092 kg-ai during 1976. Carbofuran showed an overall presence of 0.8% in water.	Water	—	<0.1 $\mu g \cdot L^{-1}$ (overall mean), 1.80 $\mu g \cdot L^{-1}$ (maximum)	Frank et al., 1982
Wascana Cr., Wood River, and Notukeu Cr. basins in southern Saskatchewan. Residue monitoring for grasshopper control program. Areas treated and quantity of carbofuran used not given. (Sampling conducted on July 4, 1985.)	Water	0/8	Not detected with 0.5-$\mu g \cdot L^{-1}$ detection limit	Ferris, 1985
Assiniboine River, Carrot River, Whitefox River, and Melfort Cr. basins in southern Saskatchewan. Residue monitoring for wheat midge control program. Areas treated and quantity of carbofuran used not given. (Sampling conducted on July 23, 1985.)	Water	0/8	Not detected with 0.5-$\mu g \cdot L^{-1}$ detection limit	Ferris, 1985
Small agriculture watershed, Black Cr., Allen County, northeastern Indiana.	Water	0/7	Not detected with 0.1-$mg \cdot L^{-1}$ detection limit	Dudley and Karr, 1980
No indication of amount of carbofuran. (Sampling conducted in July 1977.)	Sediment	0/14	Not detected with 0.1-$mg \cdot kg^{-1}$ detection limit	
	Fish	0/18	Not detected with 0.1 $mg.kg^{-1}$ detection limit	

*In 1973 each pit was sampled before the first runoff of the growing season, immediately after the first runoff, at midstream, in late summer, and in the autumn. In 1974, each pit was sampled in May, June, July, August, and November without regard to runoff.

Table A-1. Continued

Location and conditions	Matrix	Samples with carbofuran/ samples collected	Concentration	Reference
9 impoundments in the Saint John (New Brunswick) River basin. Carbofuran use in basin not given. (Sampling conducted in September/October 1983.)	Water	5/14	0.03 to 0.06 $\mu g \cdot L^{-1}$	Bailey, 1985
	Sediment	0/27	Not detected with 0.01-$\mu g \cdot g^{-1}$ detection limit	
84 sites in Atlantic provinces over a 3-year period (1979–1981).	Water	0/21	Detection limit not given	Bailey and Howell, 1983
	Sediment	0/21	Detection limit not given	
14 sampling sites (6 in a lake, 3 in a river, and 5 in a connecting canal) in the northwestern portion of Greece. Use of carbofuran in the area in 1984–1985 = 2800 kg-ai. (Sampling conducted from September 1984 to September 1985.)	Lake water	22/26	6.05 $ng \cdot L^{-1}$ (mean), 42 $ng \cdot L^{-1}$ (maximum)	Albanis et al., 1986
	Canal water	5/10	7.8 $ng \cdot L^{-1}$ (mean), 21 $ng \cdot L^{-1}$ (maximum)	
	River water	12/15	4.1 $ng \cdot L^{-1}$ (mean), 14 $ng \cdot L^{-1}$ (maximum)	
Loblolly pine seed orchard, adjacent to Lake Oconee, central Georgia. Carbofuran (granular) applied beneath soil with seed drill set for 5-cm depth. Applications in late winter or early spring of 19 $kg\text{-}ai \cdot ha^{-1}$ (Fish sampling conducted monthly or bimonthly from February 1981 to September 1984.)	Stormwater runoff 1981	-/-	Not detectable to 7820 $\mu g \cdot L^{-1}$	Bush et al., 1986
	Fish tissue (whole fish) 1981	1/28	1390 $\mu g \cdot kg^{-1}$ (as 3-hydroxy carbofuran)	
	Fish tissue (whole fish) 1982	2/29	310 and 560 $\mu g \cdot kg^{-1}$	Bush et al., 1986
		2/17	790 and 1490 $\mu g \cdot kg^{-1}$ (as 3-hydroxy carbofuran)	
	1983	0/19	Not detected with 20-$\mu g \cdot kg^{-1}$ detection limit	
	1984	0/19	Not detected with 20-$\mu g \cdot kg^{-1}$ detection limit	
2 wells on a farm near Portage la Prairie, Manitoba	Groundwater	8/14	Detected in the 11.5- to 158.4-$\mu g \cdot L^{-1}$ range in 1982	Krawchuk and Webster, 1987
			Detected in the <0.5- to 1.0-$\mu g \cdot L^{-1}$ range in 1983	
Unspecified sites in Maryland, Wisconsin, and New York. (Sampling dates not given.)	Groundwater	-/-	Detected in the 1- to 50-$\mu g \cdot L^{-1}$ range	Cohen et al., 1984, 1986

Table A-1. Continued

Location and conditions	Matrix	Samples with carbofuran/ samples collected	Concentration	Reference
31 sites (6 in New Brunswick, 10 in Nova Scotia, 7 in Prince Edward Island, and 8 in Newfoundland) were sampled for carbofuran in sediments. (Sampling conducted in 1982.)	Sediment	0/26	Not detected with 0.01-$\mu g \cdot kg^{-1}$ detection limit	Bailey, 1984
4 sites in the northeastern United States (West Lafayette, Indiana; Potsdam, New York; Tiffin, Ohio; Parsons, West Virginia) during 1985.	Rainwater	-/-	Detected in the <0.1- to 0.5-$\mu g \cdot L^{-1}$ range	Richards et al., 1987

Appendix B
Carbofuran Acute Toxicity Values for Aquatic Organisms

Table B-1. Carbofuran Acute Toxicity Values for Aquatic Organisms

Species	Test conditions	LC_{50}/EC_{50} ($mg \cdot L^{-1}$) 24 h	48 h	96 h	Formulation (% active ingredient)	Temp. (°C)	pH	Hardness (mg $CaCO_3 \cdot L^{-1}$)	Reference
VERTEBRATES									
Coho salmon (*Oncorhynchus kisutch*)	S, U	0.53 (0.43–0.65)			Technical material (99%)	12.0	7.5	44	Mayer and Ellersieck, 1986
Rainbow trout (*Salmo gairdneri*)	S, U	0.68 (0.54–0.86)			Technical material (99%)	12.0	7.4	44	Mayer and Ellersieck, 1986
Rainbow trout (*Salmo gairdneri*) (Steelhead strain)	S, U	1.02 (0.64–1.64)		0.60 (0.44–0.83)	Technical material (99%)	12.0	7.5	44	Mayer and Ellersieck, 1986
Brown trout (*Salmo trutta*)	S, U	0.84 (0.71–1.01)			Technical material (99%)	12.0	7.5	44	Mayer and Ellersieck, 1986
Brown trout (*Salmo trutta*)	FT, U	0.36 (0.24–0.52)		0.28 (0.20–0.38)	Technical material (99%)	12.0	7.5	314	Mayer and Ellersieck, 1986
Lake trout (*Salvelinus namaycush*)	FT, U	0.16 (0.12–0.23)			Technical material (99%)	12.0	7.5	314	Mayer and Ellersieck, 1986
Fathead minnow (*Pimephales promelas*)	S, U	2.24 (1.59–3.15)		1.99 (1.39–2.86)	Technical material (99%)	12.0	7.5	44	Mayer and Ellersieck, 1986
Fathead minnow (*Pimephales promelas*)	S, U	0.88 (0.49–1.58)			Technical material (99%)	17.0	7.1	44	Mayer and Ellersieck, 1986
Fathead minnow (*Pimephales promelas*)	FT, U	1.32 (0.99–1.76)		1.18 (0.81–1.71)	Technical material (99%)	17.0	7.5	314	Mayer and Ellersieck, 1986

S = static
FT = flow-through
M = concentrations in test water measured
U = concentrations in test water not measured
EC = effective concentration

Table B-1. Continued

Species	Test conditions	LC_{50}/EC_{50} ($mg \cdot L^{-1}$) 24 h	48 h	96 h	Formulation (% active ingredient)	Temp. (°C)	pH	Hardness ($mg\ CaCO_3 \cdot L^{-1}$)	Reference
Channel catfish (*Ictalurus punctatus*)	S, U	0.37 (0.22–0.64)			Technical material (99%)	20.0	7.4	44	Mayer and Ellersieck, 1986
Bluegill (*Lepomis macrochirus*)	S, U	0.37 (0.29–0.47)			Wettable powder (50%)	18.0	7.1	44	Mayer and Ellersieck, 1986
Bluegill (*Lepomis macrochirus*)	S, U	0.10 (0.09–0.12)		0.088 (0.08–0.10)	Technical material (100%)	17.0	7.1	40	Mayer and Ellersieck, 1986
Yellow perch (*Perca flavescens*)	S, U	0.28 (0.23–0.34)		0.24 (0.21–0.28)	Technical material (99%)	12.0	7.5	44	Mayer and Ellersieck, 1986
Yellow perch (*Perca flavescens*)	S, U			0.12 (0.08–0.18)	Technical material (99%)	12.0	9.5	44	Mayer and Ellersieck, 1986
Yellow perch (*Perca flavescens*)	S, U	0.54 (0.43–0.69)		0.40 (0.29–0.55)	Technical material (99%)	12.0	7.5	42	Mayer and Ellersieck, 1986
Coho salmon (*Oncorhynchus kisutch*)	S, U			0.53 (0.43–0.65)	Technical material (99%)	12	7.2–7.5	40–50	Johnson and Finley, 1980
Rainbow trout (*Salmo gairdneri*)	S, U			0.53 (0.43–0.65)	Technical material (99%)	12	7.2–7.5	40–50	Johnson and Finley, 1980
Brown trout (*Salmo trutta*)	S, U			0.56 (0.48–0.66)	Technical material (99%)	12	7.2–7.5	40–50	Johnson and Finley, 1980
Lake trout (*Salvelinus namaycush*)	FT, U			0.16 (0.12–0.23)	Technical material (99%)	12	7.2–7.5	40–50	Johnson and Finley, 1980
Fathead minnow (*Pimephales promelas*)	S, U			0.87 (0.48–1.59)	Technical material (99%)	17	7.2–7.5	40–50	Johnson and Finley, 1980

Table B-1. Continued

Species	Test conditions	LC_{50}/EC_{50} ($mg \cdot L^{-1}$) 24 h	48 h	96 h	Formulation (% active ingredient)	Temp. (°C)	pH	Hardness (mg $CaCO_3 \cdot L^{-1}$)	Reference
Channel catfish (*Ictalurus punctatus*)	S, U			0.25 (0.094–0.65)	Technical material (99%)	20	7.2–7.5	40–50	Johnson and Finley, 1980
Mosquito fish (*Gambusia affinis*)	S, U	0.96 (0.74–1.36)	0.57 (0.44–0.66)	0.52 (0.45–0.57)	EC (40%)	Not given	Not given	Not given	Davey et al., 1976
Green sunfish (*Lepomis cyanellus*)	S, U	0.31 (0.22–0.36)	0.17 (0.05–0.23)	0.16 (0.10–0.21)	EC (40%)	Not given	Not given	Not given	Davey et al., 1976
Bluegill (*Lepomis macrochirus*)	Not given			0.12	Not given	Not given	Not given	Not given	Frank et al., 1982
Yellow perch (*Perca flavescens*)	S, U			0.15 (0.12–0.19)	Technical material (99%)	12	7.2–7.5	40–50	Johnson and Finley, 1980
Bluegill (*Lepomis macrochirus*)	S, U			0.24 (0.19–0.31)	Wettable powder (50%)	18	7.2–7.5	40–50	Johnson and Finley, 1980
Singii (*Saccobranchus fossilis*)	S, U	0.649	0.603	0.547 (0.47–0.64)	Not given (75%)	18.2±2	7.2±0.2	Not given	Verma et al., 1982
Tilapia (*Tilapia mossambica*)	S, U			0.44	Not given	27.9±0.84	7.0	Not given	Konar and Ghosh, 1983
Sheepshead minnow (*Cyprinodon variegatus*)	FT, M			0.386	Not given	20	Not given	Not given	Hansen and Parrish, 1977
Indian carp (*Labeo rohita*)	S, U			4.8	Not given	27±2	7.2	60–70	Kulshrestha et al., 1986
Indian carp (*Catla catla*)	S, U			5.1	Not given	27±2	7.2	60–70	Kulshrestha et al., 1986
Indian carp (*Cirrhinus mrigala*)	S, U			4.7	Not given	27±2	7.2	60–70	Kulshrestha et al., 1986
Air-breathing catfish (*Clarias batrachus*)	Not given			1.8	Not given	Not given	Not given	Not given	Sadhu and Mukhopadhyay, 1985

Table B-1. Continued

Species	Test conditions	LC_{50}/EC_{50} (mg·L^{-1}) 24 h	48 h	96 h	Formulation (% active ingredient)	Temp. (°C)	pH	Hardness (mg $CaCO_3$ ·L^{-1})	Reference
African catfish (*Mystus vittatus*)	S, U			0.31	Wettable powder (75%)	Not given	Not given	Not given	Verma et al., 1980
Frog (tadpole) (*Micropyia ornata*)	S, U			13.47 (8.55–21.21)	Not given (75%)	25±2	7.3±0.1	55±5	Pawar and Katdare, 1983b
European carp (*Cyprinus carpio*)	S, U			0.16	Carbofuran 3G (3%)	27±2	7.2	60–70	Kulshrestha and Arora, 1986
Rainbow trout (*Salmo gairdneri*)	Not given		8.5		Furadan (5%)	Not given	Not given	Not given	Hejduk and Svobodova, 1980
European carp (*Cyprinus carpio*)	Not given		11.0		Furadan (5%)	Not given	Not given	Not given	Hejduk and Svobodova, 1980
Guppy (*Poecilia reticulata*)	Not given		3.4		Furadan (5%)	Not given	Not given	Not given	Hejduk and Svobodova, 1980
Freshwater teleost (*Ophiocephalus punctatus*)	S, U	0.32 0.30	0.27 0.26	0.18 0.21	Not given (100%)	18.2	6.9–7.4	Not given	Verma, Rani, et al., 1981
African catfish (*Mystus vittatus*)	S, U	0.50 0.46	0.44 0.37	0.31 0.26	Not given (100%)	18.2	6.9–7.4	Not given	Verma, Rani, et al., 1981
INVERTEBRATES									
Ostracod (*Heterocypris iuzonensis*)	S, U		2.4 (1.4–4.0)		Not given (100%)	20	8.2	Not given	Grant et al., 1983
Ostracod (*Cyprinotus carolinensis*)	S, U		0.40 (0.34–0.56)		Not given (100%)	20	8.2	Not given	Grant et al., 1983
Cladoceran (*Daphnia pulex*) (without suspended) sediment)	S, U		0.035 (0.027–0.046)		Furadan (40.6%)	15	7.6	282	Hartman and Martin, 1985

Table B-1. Continued

Species	Test conditions	LC_{50}/EC_{50} (mg·L^{-1}) 24 h	48 h	96 h	Formulation (% active ingredient)	Temp. (°C)	pH	Hardness (mg $CaCO_3$ ·L^{-1})	Reference
Cladoceran (*Daphnia pulex*)	S, U (with suspended sediment conc. not given)		0.045 (0.033–0.061)		Furadan (40.6%)	15	7.6	282	Hartman and Martin, 1985
Cladoceran (*Cyclops viridis*)	S, U		0.62		Not given (75%)	27.9±0.84	7.0	Not given	Konar and Ghosh, 1983
Oligochaete (*Brachiura sowerbyi*)	S, U		7.20		Not given (75%)	27.9±0.84	7.0	Not given	Konar and Ghosh, 1983
Prawn (*Macrobrachium kistnensis*)	S, U			0.157 (0.115–0.216)	Technical grade (75%)	28±2	7.4±0.2	50±2	Pawar and Kaldare, 1983a
Oligochaete (*Tubifex tubifex*)	S, U	20	18	14	Furadan (3%)	18±0.3	8.15±0.3	165±5	Dad et al., 1982
Oligochaete (*Limnodrilus hoffmeisteri*)	S, U	16	14	11	Furadan (3%)	18±0.3	8.15±0.3	165±5	Dad et al., 1982
Cladoceran (*Daphnia magna*)	S, U		0.048 (0.035–0.064)		Technical grade (99%)	20±1	7.2	60	Johnson, 1986
Midge (*Chironomus riparius*)	S, U		0.056 (0.031–0.099)		Technical grade (99%)	20±1	7.2	60	Johnson, 1986
Midge (*Chironomus tentans*)	S, U	0.0016			(at least 90%)	22°C	Not given	Not given	Karnak and Collins, 1974